PRACTICE OF MEDICINE INFECTIONS

Dr. Sunila MD (Hom)

Medical Officer, Department of Homeopathy Govt of Kerala

Infection: Lodging & multiplication of the organisms in or on the tissues of host. **Primary infection:** Initial infection of a host by a parasite.

Reinfection: Subsequent infections by the same parasite in the same host.

Secondary infection: Infection by another organism in a person suffering from an infectious disease. **Nosocomial infection:** Cross infections occurring in hospitals.

Superinfections: Infections caused by a commensal bacterium in patients who receive intensive chemotherapy.

Opportunistic infections: Organisms that ordinarily do not cause disease in healthy persons may affect individuals with diminished resistance.

Latent infections: When a pathogen remains in a tissue without producing any disease, but leads to disease when the host resistance is lowered.

• Commonest infective disease: common cold.

PYREXIA OF UNKNOWN ORIGION (PUO)

When the temperature is raised **above 38.3**°C for more than 2 weeks without the cause being detected by physical examination or laboratory tests \rightarrow PUO (FUO)

<u>Etiology</u>

- *a)* Occult tuberculosis
- b) Chronic suppurative lesions of the liver, pelvic organs, urinary tract, peritoneum, gall bladder, brain, lungs, bones & joints & dental sepsis (occasionally).
- c) Viral infections:
 - Viral hepatitis
 - Infectious mononucleosis
 - > Cytomegalovirus infection
 - Aids
- d) Connective tissue disorders:
 - > Giant cell arteritis.
 - > RA
 - Rheumatic fever
 - > SLE
 - > **P**AN (polyarteritis nodosa)
- e) Chronic infections:
 - Syphilis
 - Hepatic amoebiasis
 - Cirrhosis liver
 - Malaria
 - Filariasis
 - Leprosy
 - Brucellosis
 - Sarcoidosis
- f) Haematological malignancies
 - > Leukemia
 - Lymphoma
- Multiple myeloma
 g) Other malignant lesions: Tumours of lungs, kidney etc.

h) Allergic conditions

- i) *Miscellaneous conditions:* Hemolytic anaemia, dehydration in infants etc.
- j) Factitious fever: Self induced fever in patients with psychological abnormalities..

SEPTIC SHOCK

- Endotoxines of gram negative bacilli are responsible for most of the cases.
- More frequent in *men*.
- **Toxic shock syndrome in women**: caused by *toxigenic strains of staphylococci* (gram positive) contaminating vaginal tampons.

Clinical features:

- Features of shock: Hypotension, Weak thready pulse, Cold clammy skin, Tachycardia & Peripheral cyanosis.
- Death is caused by: *pulmonary oedema, tissue anoxia, cardiac arrythmias* & *Disseminated intravascular coagulation*

SYSTEMIC DISEASES CAUSED BY COCCI

STREPTOCOCCAL INFECTIONS

Majority of pathogenic strains are: beta haemolytic which possess streptolysin O & S

Streptococcal pharyngitis	I. P: 2-4 days	
	I. P: 1-3 days	
Scarlet fever	Primary lesion is in throat. Erythematous rash on 2 nd day over the	
	neck & trunk sparing palms & soles.	
	Diagnosis: isolation of group A streptococci from exudates.	
	Acute spreading infection of skin & subcutaneous tissue.	
Erysipelas		
	Face is commonly affected.	
Pyoderma/ streptococcal	Deeply ulcerated impetigo: ecthyma	
impetigo		
Cellulitis	Spreading inflammation of subcutaneous tissue due to entry of	
	organism through the abrasions of the skin.	
Lymphangitis	Linear red streaks from the site of entry to draining lymph nodes	
Streptococcal bacteremia	a Toxic shock syndrome may be produced by streptococci.	
Necrotizing fasciitis/	5/ Necrosis of fascia & adipose tissue, often sparing the skin.	
streptococcal gangrene		
Streptococcal myositis	Infection reaches the muscles through the bloodstream.	
Pneumonia & empyema	Streptococcal pneumonia→ bronchopneumonia	

ACUTE RHEUMATIC FEVER

Cause:

Hypersensitivity reaction to *group A streptococci*. Rheumatic fever follows 2-3 weeks after an attack of *streptococcal pharyngitis*.

Age group: 5-15 years (mean age- 6 years) Pathology: 2 stages:

- Exudative stage: acute phase.
- > Proliferative stage: prolonged process. Hallmark of proliferative phase is **Aschoff bodies.**

Heart: Endocarditis, myocarditis & pericarditis (pancarditis). **MacCallum's patch** is seen in posterior wall of left atrium due to *scarring of mural endocarditis*.

Pericardium: **bread & butter appearance** (fibrinous inflammation in pericardium).

Joints: Acute synovitis.

<u>Clinical features</u> JONES CRITERIA (revised)

Major manifestations	Minor manifestations
1. Carditis	i) Previous rheumatic fever or H/o rheumatic heart disease.
2. Polyarthritis	

* [, nor fun	Jaccoud's arthritis: affect the hand in 50% cases; n- erosive joint deformities with preservation of hand ction]	ii) Fever
3.	<i>Chorea</i> (Syn: Sydenham's chorea, St. Vitus dance, minor chorea) → non- repetitive, rapid & jerky involuntary movements of distal joints; mostly upper limbs.	 iii) Acute phase reactants → lab tests helpful in acute phase. ESR -↑, C-Reactive protein -↑
4.	<i>Erythema marginatum</i> →non- pruritic.	
5.	Subcutaneous nodules	iv) Prolongation of PR interval in ECG.
6.	Evidence of preceding streptococcal infection (ASO titer)	

<u>Diagnosis</u>

- > Throat swab culture.
- Serodiagnosis
 - Anti-streptolysin O titer (ASO)
 - Anti-streptokinase (ASK)
 - AntiDNAse B
 - Anti- nicotinamide- adenine dinucleotidase (anti- NADase)
 - Anti- hyaluronidase (AH)
 - Anti- streptozyme test (ASTZ)
- > Acute phase reactants \rightarrow lab tests helpful in acute phase.
 - ESR & C reactive protein increased.
- ➤ ECG:
 - Sinus tachycardia, ectopic beats & 1st & 2nd degree heart blocks.
 - ST elevation \rightarrow pericarditis.

POSTSTREPTOCOCCAL GLOMERULONEPHRITIS (GN)

- > This may follow either *cutaneous or pharyngeal lesion by group A streptococcus.*
- > 10- 15% of children getting recurrent skin infections may develop glomerulonephritis.
- Serotypes 12, 44, 2, 52, 55, 57 & 4 are more often nephritogenic.
- > Latent period for the development of acute GN is 10 days after pharyngitis & 3weeks after pyoderma.

STAPHYLOCOCCAL INFECTIONS

SUPERFICIAL LESIONS

Furuncle	Acute necrotic infection of hair follicle.	
Carbuncle	Large furuncle or an aggregate of interconnected	
	furuncles.	
	Sites: back of neck, hips & thighs.	
	Common in <i>diabetics.</i>	
Impetigo	Bullous impetigo is purely staphylococcal (occurs in	
	newborns & infants)	
Ecthyma	Sites: buttocks, thighs & legs.	
Sycosis barbae	Seen in <i>males after puberty</i> on the <i>beard</i> region.	
Follicular impetigo of Bockhart	Infection of hair follicle in scalp (seen in	
	childhood).	
The scalded skin syndrome (Syn: Pemphigus	s Generalized exfoliative dermatitis caused by <i>Staph</i> .	
neonatorum, Ritter's disease, toxic epidermal	al Aureus.	
necrolysis)		
Staphylococcal pneumonia	Carries a higher mortality especially in old &	
	debilitated patients.	
Osteomylelitis	Primary osteomyelitis	
Staphylococcal bacteremia	Fulminent sepsis may lead to death within 24	
	hours.	
Staphylococcal food poisoining	Symptoms start within 6-8 hrs.	
	Gastroenteritis or dysenteric symptoms.	

Tropical promyositis	Large abscess in the muscles of limbs or trunk in apparently healthy adults.		
Toxic shock syndrome	Caused by a <i>diffusible toxin produced by staphylococci (phage Type 1)</i>		

PNEUMOCOCCAL INFECTIONS

Pneumococcal Pneumonia (Syn: Lobar pneumonia)

Most common pneumococcal lesion in adults is *pneumonia*.

Pathology: Formation of inflammatory exudate in alveoli.

Stages: Red hepatisation (alveoli are filled with red cells & fibrin), Grey hepatisation (neutrophil leucocytes predominate) & Resolution.

Clinical features

- > Fever, chills & herpes simplex.
- > Pleuritic pain & Cough with pinkish or rusty sputum
- Signs of consolidation over the affected lobe.

Laboratory findings

- Rusty sputum
- Pneumococci can be demonstrated by gram staining, Blood culture is positive in 20-25% cases in early stage of the disease. Leucocytosis
- > Skiagram of the chest shows a homogenous opacity corresponding to the lobe involved.

Complications

- Local: atelectasis, lung abscess, delayed resolution
- > Due to spread of inflammation to adjacent structures: pleural effusion, empyema, pericarditis & peritonitis
- > Haematogenous spread: septicaemia, meningitis etc.

≻

MENINGOCOCCAL INFECTIONS

Neisseria Meningitidis (meningococcus, gram negative) is pathogenic exclusively to man.

Major lesions are CERTEBROSPINAL MENINGITIS & MENINGOCOCCAL SEPTICAEMIA

	Age: 6 months- adolescence. I.P: 3-5 days		
	Clinical features		
	Fever & other constitutional features like headache, neck rigidity etc.		
	Signs of meningeal irritation		
	Kernig's sign: pain over the hamstrings when the knee is extended passively with the hip flexed to 90°, & the extension is restricted.		
Meningococcal meningitis	sign is elicited		
(cerebrospinal fever)	 Brudizinski's neck sign: flexion of both legs when the neck is 		
	passively flexed.		
	Rise in intra cranial tension can cause papilloedema, blindness, deafness,		
	hemiplegia & coma.		
Meningococcal	Fulminent or chronic.		
septicemia	Schwartzman phenomenon: vascular damage occurs as an allergic		
(meningococcemia)	phenomenon caused by the endotoxin of meningococci.		
	Waterhouse- Friderichsen syndrome: haemorrhage into adrenal gland		
	result in acute adrenal failure producing toxaemia & shock.		
	Diagnosis: clinical features, lumbar puncture.		
	CSF is under pressure, turbid & shows large number of polymorphs (1000-		
	10000/cmm) containing intracellular gram negative diplococci.		

COMMON BACTRIAL INFECTIONS OF CHILDHOOD

Disease	Agent	Clinical features	Complications	Diagnosis &
Diphtheria	Corynebacterium diphtheriae, gram +ve. 3 types: • Gravis • Intermedius • Mitis Virulent strains produce exotoxin.	I.P:3-4 days Pharyngeal diphtheria: Most common. Bull neck (gross cervical lymhadenopathy) Malignant diphtheria (oedema of submandibular area) Laryngeal diphtheria: Produces respiratory obstruction early. Nasal diphtheria Cutaneous diphtheria: punched out ulcer	Mechanical obstruction of the airways. Toxic complications: most pronounced in the heart & motor nerves. 3 rd , 6 th , 7 th , 9 th &10 th cranial nerves are commonly affected.	prevention. Demonstration of organism in stained smears made from the membrane & by culture using Loeffler's medium. Fluorescent antitoxin staining- rapid diagnosis. Toxigenicity is assessed by: o Guine a a pig inoculation Passi ve agar gel diffusion (Elek plate method) o Coun ter immunoela
Whooping cough/ pertussis.	Bordetella pertussis. Bordetella parapertussis. Bordetella bronchiseptica.	Incidence: children below 5 years. I.P: 7-14 days. Course: 6-8 weeks. <u>3 stages:</u> Catarrhal stage: most infective stage. Paroxysmal stage: cough starts, increase in severity & becomes repetitive & explosive. Each paroxysm is followed by a whoop (inspiratory effort through a narrowed glottis). Convalescence stage.	 A. Respiratory: Bronchitis Bronchopneumonia Atelectasis Emphysema or pneumothorax. Bronchiectasis Flare- up of tuberculasis. B. CNS: Convulsions C.GIT: Severe vomiting with dehydration. Tetany Prolapse of rectum Hernia D. Haemorrhages E. Malnutrition 	immunoele ctrophoresi s Prevention: DPT. • Clinical features • WBC count elevated (20000 to 50000/ cmm) • Absolute lymphocy tosis • Chest-X- Ray: perihilar infiltrates or segmenta I collapse. • Culture: nasophar yngeal swab. • Fluoresce nt antibody staining:

				rapid &specific diagnosis. Prevention: DPT (killed B pertussis). 1 st dose at 2 morths, 2 more doses at 4 week interval
Haemophilus influenzae infections	Haemophilus influenzae (gram – ve). 6 types: a -f based on capsular polysaccharide. PRP (polyribosyl ribitol phosphate) determines its virulence.	Frequently cause pharyngitis in children.	Secondary infection by H. influenzae causes bronchopneumonia & exacerbation of chronic bronchitis.	Gram staining & Culture, CSF, sputum etc. (Organism is highly susceptible to low temperatures. So refrigeration during transit should be avoided. Prevention: Hib polysacchari de tetanus protein conjugate vaccine/PRP -T) 2 nd , 3 rd & 4 th months; booster dose: after 2 years.

OTHER BACTERIAL INFECTIONS.

TYPHOID FEVER

Immunity: one attack confers *life long immunity*, but 2nd attack occurs rarely.

Epidemiology	Agent: Salmonella typhi; spread: fecal- oral route through contaminated food, water, ice creams, milk etc. Infective dose: 10 ⁷ organisms. Healthy carriers: fecal carriers or urinary carriers. Chronic asymptomatic carrier state may be present with persistent infections in the gall bladder .		
Pathogenesis	 Factors determining the establishment of infection: 1. Size of the inoculum; larger the dose, chance of infection ↑ 2. Normal gastric acid kills the bacilli. 3. Virulence of the infecting strain 4. Presence of bacterial flora in the jejunum. 		
	Organ involved Clinical lesion		
Pathology	1. MeningesTyphoid meningitis2. LungsTyphoid pneumonia3. Gall bladderCholecystitis, Cholelithiasis4. BonesOsteomyleitis, bone abscesses (Brodie's abscess)5. Heart valves, Intravascular prosthesisSalmonella endocarditis		
	1 st week: temperature rises in <i>a step ladder pattern. Rose spots</i> (due to bacterial embolism) over upper abdomen & chest; slow pulse fever; constipation & less often		

	diarrhea, abdominal pain etc.			
Clinical				
features				
	2 nd week:	temp. Continuous (40°C); Tongue coated in the centre, margins reddish		
	(enteric feve	ver)		
	<i>c</i> : <i>c i</i>			
	Signs of tox	aemia: muttering delirium; subsuitus tendinum (tremulousness of the		
	tumidity (a	hdomen moderately distended) especially over <i>right iliac fossa</i>		
	(a	buomen moderately distendedy especially over right macrossa.		
	3 rd week:	fever & toxemia continues. Complications develop during 3 rd week. In		
	uncomplicat	ed cases the temperature starts to fall by lysis at the end of the third		
	week & beco	pmes normal within 1 week.		
	General	Toxemia & typhoid state, DIC, circulatory collapse, dehydration, relapse		
Commiliantian a	017	etc.		
Complications	GII	Abdominal distention, diarrnea, perforation of the intestine (site:		
	CNS	Coma meningism meningitis peripheral neuritis deafness		
	Skin	Bed sores alonecia		
	Others	Myocarditis pyelonenhritis alomerulonenhritis osteomyelitis arthritis		
Diagnosis	1. WBC co	punt: 1, relative lymphocytosis		
	2. Isolatio	on of the organism from blood, urine or feces		
	3. Feces 8	& urine culture become +ve in 2 nd & 3 rd week of illness.		
	4. Typhoid meningitis: CSF contains the organism			
	5. bone marrow culture			
	6. <u>Widal test:</u> This test detects & measures the <i>H</i> & <i>O</i> agglutinins of typhoid &			
	paraty	phola bacilli in the patient's serum. The antibody titers increase steadily		
	after the first week till the 4 th week & decline. A 4 fold rise in the titer of O			
		s specific than Ω antibodies. The Ω and utining are of greater value in		
	diagno	sis & titer of 1: 200 or more is very suggestive.		
	be less specific than O antibodies. The <i>O agglutinins</i> are of greater value in diagnosis & titer of <i>1: 200 or more</i> is very suggestive.			

PARATYPHOID FEVERS

- Agent: Salmonella paratyphi A, B, C.
- Illness resembles typhoid, though the *toxemia* & *complications are milder*.

SHIGELLA INFECTIONS (BACTERIAL DYSENTERY)

Shigellae: 4 species

- > Shigella dysentriae type 1 & 2(S. shigae & S. schimitzii)
- S. flexneri.
- > S. sonnei.
- ➤ S. boydii.

Source: contaminated food or water.

Pathology

The *entire colon* may be affected. Toxins are responsible for secretory diarrhea. Necrosed mucosa \rightarrow *intestinal casts.*

Differentiating features between bacillary & amoebic dysentery

Clinical features	Bacillary dysentery	Amoebic dysentery
onset	Sudden	Slow
I. Period	A few days	Weeks to months
Number of stools in 24 hours	Over 20	Below 15
Nature of feces	Mucus & blood	Fecal matter mixed with mucus & blood
Reaction of feces	Alkaline	Acid
Clinical course	acute	Sub acute & recurrent
Microscopy of feces	Cellular exudates	Erythrocyte & Entamoeba histolytica.
Culture	Shigella isolated	Special methods to culture amoeba.

Complications

- Dehydration
- Electrolyte imbalance
- Circulatory failure
- Renal failure
- > Intestinal perforation & rectal bleeding
- Paralytic ileus.

ANTHRAX (Wool Sorter's disease)

Agent: Bacillus anthracis (gram +ve)

Source: infected animals, contaminated wool, hair etc.

Clinical features

- 1. Cutaneous form (malignant pustule)
- 2. Pulmonary form: anthrax pneumonia (wool sorter's disease)
- 3. Gastrointestinal form
- 4. Meningitic form

Diagnosis

- > Blood Culture: *polymorph leucocytosis*
- > CSF: haemorrhagic in anthrax meningitis
- > Agar gel precipitation inhibition test: a four fold rise in serum antibody titer confirms the diagnosis.

Klebsiella pneumoniae: Freidlander's bacillus

Pseudomonas aeruginosa: produces a greenish pigment→ pycocyanin

Proteus: 4 species: P. mirabilis, P. vulgaris, P. morganii, and P. rettgeri.

They cause super infection in areas of tissue damage (eg. umbilical stump in newborn \rightarrow bacteremia & septic meningitis)

ESCHERICHIA COLI INFECTIONS

Group: Enterobacteriaceae. Gram negative

Antigens:

- somatic (0)
- Flagellar (H)
- Capsular(K)

Clinical presentation

- > UTI
- Peritonitis
- Septicemia
- Neonatal infection
- Biliary tract disease
- Gastroenteritis:
 - Enterotoxigenic strains of E. coli (ETEC) cause gastroenteritis in children's nurseries.
 - Traveller's diarrhea

HAEMOLYTIC URAEMIC SYNDROME

- Coli serotypes 015, K7, and H7 cause haemorrhagic colitis.
- ♦ Watery diarrhoea→ bloody.
- Verocytotoxines are responsible for haemolytic uraemic syndrome

PLAGUE

- Zoonosis.
- Causative agent: **Yersinia pestis** (previously known as Pasteurella pestis); gram negative.
- Vector: Xenopsylla cheopis; infected rat flea.
- Reservoir of infection: infected domestic rodents such as Rattus norwegius, Rattus rattus & Mus musculus.

Pathogenesis: The organisms enter through the skin. The bacilli reach the local lymph nodes which enlarge & suppurate: Bubo. Bacilli proliferate and enter the blood stream to produce metastatic lesions. Spleen may be enlarged twice or thrice its normal size. In primary pneumonic plague, the organisms reach the lungs through the respiratory tract.

* Clinical manifestations:

- Bubonic plague:
 - a. I. P: 2-6 Days.
 - b. Fever & lymphadenopathy
 - C. *Buboes:* in inguinal & axillary region.
 - d. *Pestis minor:* milder cases seen during epidemics. These present only with buboes.
- Septicemic Plague:
 - a. Chills, fever, tachycardia, headache, vomiting and delirium
 - b. Death may occur with in a few days before localizing lesions are evident.
 - c. Haemorrhagic manifestations may develop.
- Pneumonic Plague: Primary and Secondary
 - a. *Primary:* More fulminant and rapidly fatal. Organisms reach the lungs by inhalation.
 - b. *Secondary:* Organisms reach the lungs through the blood stream

Diagnosis:

- > Diagnosis is confirmed by demonstrating the organisms by smear, culture or animal inoculation studies.
- > *Bubo-fluid* can be aspirated and stained for the organisms.
- > Sputum can be stained for the organisms and cultured.
- > In septicemic plague *blood culture* yields the organism. There is neutrophil leukocytosis. *Specific antibodies* develop in patients *who recover from the disease.*

* Prevention:

A *heat killed vaccine (Haffkine)* is available for immunizing the population at risk.

BRUCELLOSIS (Undulent fever, Malta fever, Abortus fever)

It is an infectious disease of animals which is transmitted to man by handling infective material.

Causative organism: gram negative coccobacilli.

- Brucella abortus: causes abortion in cattle.
- Brucella melitensis: infection spreads through goat's milk
- Brucella suis: obtained from pigs.

Pathogenesis:

Organisms enter through GIT, RT, and conjunctiva or through the skin. They pass through the local lymph nodes into the blood stream to localize in the reticuloendothelial system. They multiply in RE tissues to produce granulomas.

Clinical features:

- I.P: 2-3 weeks
- Fever, malaise, *sweating*, chills, arthralgia & backache.
- Spinal tenderness, arthritis & orchitis are common.
- Chronic brucellosis may present as PUO.

Complications:

- Endocarditis
- CNS: meningoencephalitis, myelitis & polyradiculoneuropathy.

- Nephritis
- Hepatic & splenic suppuration
- Calcification in the liver & spleen & cholecystitis
- Uveitis

Diagnosis:

- Clinical features: *typhoid like illness* especially if associated with spondylitis or arthritis.
- Diagnosis is confirmed by isolation of the organism from blood, bone marrow etc.
- Special lab techniques are necessary to grow brucella.
- Standard tube agglutination tests, Coomb's antiglobulin tests, complement fixation test & ELISA test are used for detecting agglutinating antibodies. **Coomb's test** & ELISA are more reliable. If the initial test is negative, test is repeated after 3-4 weeks.

DIARRHOEAL DISEASES OF INFECTIVE ORIGIN

CHOLERA

- Causative organism: *Vibrio cholerae;* gram negative. 92 serogroups are identified based on O (somatic) antigen. The strain producing epidemic cholera possesses O1 antigen; hence this vibrio is designated as vibrio cholerae O1.
- Inaba, Ogawa & Hikojima are the most important pathogenic subtypes.
- Others are collectively designated non- O1 V. cholerae.
- *Eltor biotype* is a variant of V. cholerae O1; it is characterized by hemolytic activity & resistance to polymyxin. Differentiation between Eltor & V. cholerae is by phage typing.
- Classic disease is caused by Vibrio cholerae, but the majority of outbreaks occurring in India are due to Eltor biotype.
- Main vehicles of infection are water, cooked food kept unhygenically exposed to flies, sea foods, fruits & vegetables.

Pathogenesis:

Vibrio cholerae multiply in the jejunum & small intestine & produce an enterotoxin. By the influence of this toxin the enterocytes (intestinal mucosal cells) secrete large amount of isotonic fluid. The result is *watery diarrhea* which leads to loss of isotonic fluid. Excessive loss of fluid & electrolyte gives rise to hypovoluemic shock & metabolic acidosis.

Cholera enterotoxin:

- > It is a protein of Mol. Wt: 84000.
- > 2 immunologically distinct regions: *A* (*active*) & *B* (*binding*).
- > Binding enables A region to penetrate the mucosal cells
- > This toxin leads to formation of *adenylate cyclase* which induces excessive production of cyclic AMP (*cAMP*), which in turn is responsible for *over secretion of electrolytes* & water by the *enterocytes*.

Clinical features:

- Eltor cholera: usually *mild* & *asymptomatic*.
- Moderate & severe cases: last for 3-5 days.
 - Painless watery diarrhea & effortless vomiting (clear, watery fluid).
 - Excreta: *rice- water appearance* due to the presence of flakes of mucus & large number of vibrios.
 - Dehydration (when *severe*: sunken eyes, shriveled skin, collapsed neck veins), acidosis & shock. Thirst with dryness of mouth & tongue (earliest indication of fluid deficit); Oliguria & renal shutdown. Mental state is clear.
 - Painful muscular cramps due to hyponatremia.
 - Cold extremities.
 - Abdomen: scaphoid.
- Cholera sicca: rarely large amount of fluid may collect in the intestinal lumen & a severe dehydration, shock & death may result even before evacuation occurs.

Diagnosis:

- Isolating he organism from stool sample.
- Suitable medium for transporting specimen is: *Venkataraman & Ramakrishnan fluid or Carry & Blair medium.*

Complications:

- > Dehydration & severe shock leads to renal cortical necrosis & renal failure.
- > Hypokalemia leads to fatal cardiac arrythmias, abdominal distension & muscle paralysis.
- > Injudicious administration of electrolyte solutions intravenously without correcting metabolic acidosis may result in pulmonary oedema.
- > Convulsions in children due to cerebral venous thrombosis.
- Severe hypoglycemia.
- > Prolapse of rectum in children.
- Florid malnutrition.

Rehydration therapy: ORS (oral rehydration salt) solution.

Sodium chloride	3.5g
Sodium bicarbonate	2.5g
Potassium chloride	1.5g
Glucose	20g
Water	1 liter.

Cholera is a notifiable disease.

ROTAVIRUS DIARRHEA

- > Rotavirus affects mainly infants & young children aged 6 months to 2 years.
- > I. P: 1 to 7 days; usually less than 48 hours.
- > Vomiting occurs early & it precedes diarrhea.
- Diarrhea extends over 5-7 days but virus is shred up for 10 days.
- Breast milk may have a protective role due to the presence of maternal Ig A antibodies.
- > Rotavirus group A: major cause of endemic diarrhea in infants & young children worldwide.

Norwalk & Norwalk like agents: Cause mild gastroenteritis in school, community & family settings.

Calcivirus: Cause Rotavirus like illness in children & Norwalk like in adults.

Astrovirus: Pediatric diarrhea reported in nursing homes.

Campylobacter jejuni (vibrio fetus):

Complications of campylobacter jejuni dysentery:

- Reactive arthritis
- Guillain-Barre syndrome(GBS)

<u>Pseudomembranous colitis:</u> *syn: antibiotic associated diarrhea.*

BARTONELLOSIS (Syn: carrion' s disease, Oroya fever, Verruga Peruana)

Bartonella consists of 3 main pathogens.

- > Bartonella bacilliformis: Carrion's disease. Disease is transmitted by sandfly phlebotomous verrucarum.
- > Bartonella henselae: cat- scratch disease & bacillary angiomatosis in patients with AIDS.
- *Bartonella Quintana: trench fever.*

Clinical picture

2 distinct clinical syndromes:

- A) Oroya fever:
 - *i.* Fever, rigor, headache etc. lasts for 3-4 weeks.
 - *ii.* Organism can be demonstrated in blood smears stained by Giemsa's or Wright's strain.
 - *iii.* Death may occur due to severe anaemia or super infection salmonella.

B) Verruga Peruana:

- *i.* Hemangiomatous tumours of skin and mucous membranes
- *ii.* Mortality is very low

LEGIONNAIRE'S DISEASE

- Causative agent: Legionellaceae; gram negative; natural habitat: water
- Over 36 species; among them *L. pneumophila* is the most common human pathogen.

Clinical features:

Two main clinical syndromes

a) Legionnaire's disease

- Infection is by inhalation of aerosols
- I.P: 2-10 days
- Pneumonia is the commonest presentation followed by GIT symptoms such as watery diarrhoea

b) Pontiac fever

• Brief febrile illness resembling influenza caused by other species of legionella

Diagnosis:

 Culture of the organism from the sputum & direct fluorescent antibody staining: quick and ready method but less sensitive

ANAEROBIC INFECTIONS

TETANUS (Syn: Lockjaw)

- *Exotoxin produced by Clostridium tetani.*
- *Exotoxin: neurotoxin; tetanospasmin & hemolysin; tetanolysin.*
- * I. P: generally less than 2 weeks; it may range from 2-60 days.
- Clinical features:
 - [°] Diagnosis becomes evident when **lockjaw** sets in.
 - $^{\circ}$ **Opisthotonous:** hyperextension of the spine & neck due to rigidity and spam of back muscles.
 - ^o **Risus sardonicus:** grinning expression due to sustained contraction of facial muscles.
 - ^o Interval between first symptom & the first convulsion is called the **onset period.**
 - ^o Deep tendon reflexes are exaggerated but the plantar response is flexor.
 - ^o **Local tetanus:** symptoms confined to a part near the site of injury.
 - ^o **Cephalic tetanus:** local tetanus involves the facial muscles.
 - ^o **Tetanus neonatorum:** *tetanus occurs within 10 days of birth; inability to suck the nipple, irritability & excessive crying associated with grimacing movements of the face. Muscles of the back, neck & abdomen become spastic.*

Complications:

- ^o Respiratory obstruction & aspiration pneumonia.
- ° Hyperpyrexia
- ° Myocarditis leads to cardiac failure & hypotension.
- ° Decubitus ulcers &UTI due to prolonged immobility.

SIGNS & SYMPTOMS (COMMONEST, FIRST & MOST IMPORTANT)

Tetanus	Trismus
Brucellosis	Sweating
Acute myocarditis	Tachycardia
Impetigo & dermatophytoid	Itching is the only symptom.

GAS GANGRENE (Syn: Clostridial myonecrosis)

- Clostridium perfringens (C. welchi), C. septicum, C. novyi (C. oedematiens), C. histolyticum & C. sordelli.
- They produce local necrosis & distant lethal effects.
- I.P: 1-4 days.

ANAEROBIC FOOD POISOINING

* Clostridium perfringens food poisoning:

- ° Source of contamination: meat
- $^{\circ}~$ Heat resistant spores survive improper cooking. They germinate when the food is cooled.
- ° They produce an exotoxin which causes abdominal cramps & diarrhoea.

Clostridium botulinum produces botulism.

- ° I. P: 12- 36 hours.
- ° Fever absent & GIT symptoms are slight.

 $^{\circ}\,$ Prominent symptoms are dysphagia, diplopia, ptosis, dysarthria, muscle weakness & quadriplegia.

GENITAL SORE

Features	Syphilis (Syn: Hard chancre, Lues venerea)	Chancroid (Syn: Soft chancre, Soft Sore, Ulcus Molle)	Lymphogranuloma Venereum (Syn: Climatic bubo, lymphogranuloma Inguinale)	Granuloma Inguinale (Syn: Granuloma venereum, Donovanosis)	Herpes infection
Organism	T-pallidum	Haemophillus Ducreyi	Chlamydia Trachomalis	Calymmatobacteriu m Granulomatis (Syn: Donovania granulomatis)	HSV (Herpes Simplex Virus) type 2 & less frequently by HSV type 1.
Incubation period	9-90 days Average 21 days	3-5 days	2 weeks to several weeks	A few days to a few months.	2-7 days
Clinical features	 Painless indurated papule that may later turn on to ulcer Painless 'shotty' lymph adenopathy 	 Painful non indurated ulcer (soft chancre) Painful lymphadenop athy. Secondary infection with <i>Vincent's</i> <i>spirochetes</i> may develop. 	 Painless papule that often go unnoticed. Sometimes it may ulcerate After the genital lesion has healed painful matted suppurative lymphadenopathy Multiple sinus tracts are formed in lymph nodes Groove's sign; enlarged nodes are present below and above the inguinal ligament Vaginal and rectal strictures Elephantiasis of vulva. Esthiomene: pseudo- elephantoid condition of the genitalia caused by lymphatic obstruction. 	 Begins as one or more subcutaneous nodules that erode through skin to prduce clean granulomatous sharply defined painless lesions No lymphadenopath y 	 Painful papule that ulcerate in 3-6 days. Burning pain S₁ to S₅ dermatomes. Systemic symptoms fever, headache, malaise Inguinal lymphadeno pathy occurs
Diagnosis		Ito test	Frei's intradermal test	Donovan's	Papanicolaou stained smears
					show multinucleated giant cells with eosinophilic inclusions.

SYPHILIS

- Caused by Treponema pallidum. Ŧ
- Ŧ
- **Incubation period**: about **9-90 days** (average 21 days) T.palladium *can cross placenta at any stage of pregnancy* but lesions of congenital syphilis develop generally after 4 months of gestation with disappearance of langhans layer Ŧ (cytotrophoblast) in villi.

Lesions in syphilis

- 1. Congenital Syphilis
 - Early congenital syphilis; infantile form (within first 2 years of birth) . .
 - Late (tardive) congenital syphilis (seen after age of 2 years)
- 2. Primary Syphilis
- Secondary Syphilis 3.
- 4. Tertiary Syphilis

Early congenital syphilis (similar to secondary Late congenital syphilis acquired syphilis)

- Syphilitic snuffles (rhinitis) is earliest feature
- Vesicobullous lesion (*syphilitic pemphigus*)
- Snail track ulcers on mucosa
- . Rhagades (radiating fissures at angles of mouth)
- Condylomata
- Old man facies
- Alopecia
- Syphilitic wig: growth of black hair
- Hepatosplenomegaly
- Generalised lymphadenopathy
- Coombs negative hemolytic anaemia
- Bleeding diathesis
- Osteochondritis
- Syphilitic pseudoparalysis
- Chorioretinitis
- IUGR

Stigmata of congenital syphilis

- Hutchinson triad
 - Hutchinson teeth (peg like incisors), Interstitial keratitis & Eighth nerve deafness
- Olympian brows (frontal bossing), hot cross bun skull & Parrot nodes due to thickening of frontal & parietal bones of the skull.
- Saddle nose due to destruction of nasal bone.
- Higoumenaki's sign (U/L or B/L thickening of sternal portion of clavicle) High arched palate & bulldog facies.
- . Saber shins.

Primary syphilis

- о A small macule develops at the site of inoculation: *primary chancre*.
- ο Painless punched out non-bleeding indurated ulcer
- 0 Painless, rubbery, *shotty* lymphadenopathy

Secondary syphilis

- 0 Bilateral symmetrical, asymptomatic rashes on palms and soles
- ο Generalized non-tender lymphadenopathy
- о Condylomata lata
- 0 Moth eaten alopecia
- 0 Arthritis
- 0 Proteinuria
- **Tertiary syphilis**
 - 0 Gumma
 - ο Neurosyphilis
 - 0 General paresis

Page 14

Gumma

.

Interstitial keratitis

Bone lesions

Granulomatous periostitis & gummatous osteitis.

Visceral syphilis

Hepar lobatum: surface of the liver becomes lobular.

CNS:

GPI, taboparesis, optic atrophy etc.

Diagnostic test for syphilis

- Absolute diagnosis during the first and second stage can be made by- Direct examination, under dark ground microscopy.
- ^o Serological tests are of two types
 - Nonspecific Treponemal tests
 - Specific Treponemal antibody tests

Serological tests for syphilis	
Nonspecific Tests	Specific Test
 a) VDRL (venereal Disease Reference Laboratory test) b) Kahn test c) Complement fixation test (Wasserman Test) Non treponemal antigen tests are used for monitoring patients response to t/t, they become – ve after t/t 	 a) Flouroscent treponemal antibody- absorption (FTA- ABS) b) Treponemal pallidum hemagglution TPHA Specific treponemal tests are of little value as indicators of clinical cure, as they remain +ve in spite of t/t

- *The Most sensitive test in primary Syphilis- FTA- ABS*
- Most specific test- TPI (Treponema pallidum immobilization)> FTA- ABS
- In secondary Syphilis all tests are 100% sensitive but mostly used is VDRL
- Earliest test to become positive- FTA-ABS
- ☞ VDRL become positive after- 3-5 weeks of infection or 7-10 days of chancre.

% of patients with positive serological tests for syphilis

Test	Primary syphilis	Secondary syphilis	Tertiary syphilis
Nonspecific	50-70%	99%	70%
Specific	70-85%	100%	98%

Commonest STD: Nongonococcal urethritis

RICKETTSIAL DISEASES

Group	Agent	Vector& reservoir	Clinical features	Weil- Felix reaction
A. <u>Typhus</u> <i>i.</i> Epidemic typhus (syn: louse borne typhus, typhus exanthematicus, Gaol fever)	R.prowazekii	Body louse (man)	 I.P- 7 days Continuous fever (39-40°C) Macular skin rash appears only on the 5th day Starts to fade by about the 10th day I.P- 8-16 days 	+++ve for OX-19

<i>ii.</i> Endemic typhus (syn: <i>Murine typhus,</i> <i>flea typhus</i>)	R.typhi	Rat flea; xenopsylla cheopis (rat)	 Continuous fever for 12 days and then comes down by lysis Morbilliform rash develops in the axillae, arms, abdomen, chest, shoulders and thighs by about the 5th day Persons who suffer from typhus acquire life long immunity. Relapse occurs after a long latent period in some cases→ Brill-Zinsser disease 	+++ve for OX-19
<i>iii.</i> Brill-Zinsser disease	R.prowazekii	Body louse (man)		Usually negative
B. <u>Spotted fever</u>			Rash is prominent.	
<i>i.</i> Rocky mountain spotted fever	R. rickettsi	Ticks (rabbit, dog &rodents)	 Classic disease in this group. Rash occurs on 3rd day. 	++0X-19 and ++0X-2
 <i>ii.</i> Mediterranean fever (fever boutonneuse) <i>iii.</i> Rickettsial pox <i>iv.</i> North Asian tick borne typhus <i>v.</i> Queensland tick typhus. <i>vi.</i> Indian tick typhus <i>vii.</i> African tick bite fever 	R. conori R. akari R. siberica R. australis R. conori R. africae	Ticks (dog &rodents) Gamasid mite (mouse) Tick (wild animals, cattle, birds) Tick (bush rodents) Tick (rodents) Ticks (cattle & goat)		++OX-19 and ++OX-2 Negative +OX-19 and OX-2 +OX-19 and OX-2 +OX-19 and OX-2
C. <u>Scrub typhus</u> (Syn: Mite typhus, mite fever, Japanese river fever, Tsutsugamushi fever)	R.tsutsugamushi	Trombiculid mite (small rodents, birds)	 I.P- 6-18 days At the site of bite a necrotic ulcer (eschar) develops with local lymphadenopathy 	+++OXK
D. <u>Q fever</u>	Coxiella burneti	Ticks transmit disease in animals; human disease is occupational- cattle, sheep, goat		negative
E. <u>Trench fever</u>	R. guintana	Body louse (man)		negative

NON VENEREAL TREPONEMATOSIS

<u>YAWS</u>

- Treponema pertenue.
 Primary stage: maculopapular lesions.
- Secondary stage:
 - Crab yaws- over the palms & soles lesions become painful & walking may be restricted • due to pain.
 - Gondou- thickening of nasal bones. •
- ✤ Teritiary stage:

• Gangosa- extensive destruction of facial tissues with gross mutilation of the nose & mouth leads to the formation of a single open cavity.

PINTA: Treponema carateum.

RELAPSING FEVERS

LOUSE- BORNE RELAPSING FEVER

- Borrelia recurrentis. **
- ٠ Vector: body louse; pediculus humanus corporis.
- ٠ Allopathic drug therapy may lead to severe **Jarisch- Herxheimer reaction** with profound fall in temperature, shock & cardiac failure within a few hours.

TICK- BORNE RELAPSING FEVER

- * Borrelia duttoni.
- ٠ Vectors: soft ticks- ornithodoros tholozoni, O. crossi, O. lahorensis, O. moubata.
- ••• Jarisch- Herxheimer reaction is less pronounced.

LYME BORRELIOSIS

- ••• Borrelia burgdorferi.
- ٠ Vector: hard ticks; Ixodes ricinus, Ixodes persulcatus, I. dammini, I. pacificus & I. scapularis.
- * Stage 1: erythema migrans; rash spreaing centrifugally.
- ٠ Stage 2: early disseminated infection.
- Neurological features: Lyme Neuroborreliasis. ٠
- Meningoradiculitis: Bannawarth's syndrome: CSF pleocytosis & severe radicular pain. ٠
- ✤ Jarisch- Herxheimer reaction develops rarely.

LEPTOSPIROSIS

- Leptospira: 2 species
 - L. Interrogans ≻

0	L. icterohemorrhagiae	rat & rodents
0	L. seroje	pig

- L. seroje 0
- L. canicola dog 0
- 0 L. Pomona cattle
- L. biflexa ≻
- ٠ Leptospirae are eliminated in the *urine* of their animal hosts.
- ٠ I. P: 10 days.
- Only 5- 10 % of persons develop the clinical illness. ٠
- ٠ Male, 20-30 age group.
- Nonspecific features of viral fever. ŵ
- Conjunctival suffusion, headache, chills & jaundice. *
- ٠ 4 groups:
 - common leptospiral syndrome (CLS) 20% 0
 - CLS+ bleeding tendency 5% 0
 - CLS+ meningitis 0
 - 5% 0 CLS+ involvement of abdominal organs 70%
- $\dot{\mathbf{v}}$ Hepto- renal lesions predominate in L. icterohemorrhagiae infection (weil's disease)
- * Pretibial skin lesions predominate in L. autumnalis (Fort Bragg fever)
- ••• Meningeal symptoms prominent in L. canicola infection (canicola fever)
- Complications: Multi organ failure; renal failure, cardiac failure, respiratory failure, $\dot{\mathbf{v}}$ thromobocytopenia with bleeding etc.
- Unlike viral hepatitis SGPT are low compared to the degree of jaundice. ÷
- Leptospirae may be demonstable in urine. ٠
- Serology is positive after 1-2 weeks. Microscopic agglutination test (MAT) & indirect ٠ hemagglutination (IHA) are routinely employed.

MYCOBACTERIAL INFECTIONS

TUBERCULOSIS

Mycobacterium tuberculosis.

- T- Lymphocytes play a major role in conferring immunity against Mycobacterium tuberculosis.
- Pathology: macrophages with engulfed organisms get transformed into epitheloid cells. Langhan's giant cells are formed by the fusion of epitheloid cells. These are surrounded by lymphocytes & fibroblasts. Caseation necrosis occurs at the centre. Pathological hallmark of TB is tubercle. Granuloma formation limits the infection. Some bacilli remain in these tissues as persisters.

*** PRIMARY PULMONARY TUBERCULOSIS:**

- Initial lesion is a *sub pleural tubercle located in the upper part of lower lobe, lower part of upper lobe or middle lobe-* **Ghon's lesion**.
- Ghon's lesion with enlarged lymph nodes & interconnecting lymphangitis: *primary complex or Ghon's complex.*
- Compression of neighboring bronchi especially of middle lobe leads to collapseconsolidation & bronchiectatic changes; this may later present as *middle lobe syndrome*.
- *Fever, loss of appetite, loss of weight & cough:* early symptoms.
- Allergic manifestations like *phlyctenular conjunctivitis or erythema nodosum may herald primary TB* in some cases.
- Presentation in children includes tuberculous pneumonia, hemoptysis or asthma.
- **Physical examination:** in some, signs of pulmonary consolidation, collapse or pleural effusion.
- Tuberculin skin test (Mantoux test) is positive.
- Blood: ESR raised & lymphocytosis.
- *Chest X-ray: hilar lymphadenopathy* & pulmonary lesion may be detected by careful examination.
- * **POST PULMONARY TB (**lesion occurring in sensitized individual):
 - Pathology:
 - 1. Direct progression of primary lesion
 - 2. Reactivation of a primary focus
 - 3. Haematogenous infection of lung from lymph nodes, tonsils etc
 - 4. Reinfection or super infection.
 - Upper lobes are commonly affected.
 - 2 forms: slowly progressive nodular form & fibrocaseous form with tendency for cavitations (many of the blood vessels in the cavity are occluded by thrombosis, but those that remain patent become aneurysmal (*Rassmussen's aneurysms*).
 - Evening rise of temperature, mild & persistent cough, anorexia, loss of weight & general weakness.
 - Lesion resembles *lobar pneumonia*.

Complications of pulmonary tuberculosis

<i>Early</i> (within months)	Intermediate (within several months)	<i>Late</i> (after several years)	
Mild haemoptysis	Massive haemoptysis	Pulmonary fibrosis with compensatory emphysema & cor pulmonale.	
Pneumothorax	Secondary infection of cavities	Bronchiectasis.	
Pleural effusion	Pneumothorax, pleural effusion, empyema.	Persistence of open cavities without healing	
Poncet's syndrome	Progressive fibrosis with dyspnoea.	Aspergilloma; growth of Aspergillus fumigatus in the cavities in the form of a fungus ball.	
(allergy to TB;	Spread to other organs such as larynx.	Apical TB with carcinoma.	
polyarthritis occurring in TB.)	Non-healing of lesion.	Secondary amyloidosis.	

Sputum examination

- Most important diagnostic investigation.
- At least 3 bacilli should be seen before the smear is declared positive.

Tuberculin skin test

• Test is done by injecting PPD in the forearm intradermally. The result is read after 48 hours & the indurations is recorded (+ve: >12mm)

- Diagnostic value is in those individuals who are *recent converters*. This should suggest the occurrence of infection by mycobacteria.
- Negative results: *malnutrition, immunosuppresion therapy* &*Hodgkin's disease.*
- Patients suffering from *active TB* show an *accelerated arthus reaction*.

MILIARY TUBERCULOSIS (Syn: acute hematogenous tuberculosis)

- $^{\circ}$ Tubercle bacilli entering the blood stream are diffusely disseminated & results in military tuberculosis.
- ^o More common in *young children* in whom it is seen as a *complication of primary TB*.
- ^o Areas of *caseated vasculitis (Weigert's foci)* occurring in veins or arteries result in the discharge of bacilli into the circulation.
- ^o Majority of cases present varying grades of pyrexia.
- ^o Choroids tubercles may occur rarely but if they occur they are diagnostic.
- ^o Skiagrams of the chest shows numerous, small rounded shadows- the military mottling in upperlobes (in conditions such as pulmonary hemociderosis, eosinophilia, disseminated carcinoma, pneumoconiosis, sarcoidosis & histoplasmosis mottling is more prominent in the lower zones).

TUBERCULOUS MENINGITIS

- ^o Develops commonly as a *complication of military tuberculosis*.
- ^o Small granulomas are formed in the superficial layers of the brain (**Rich foci**).
- ^o Death occurs in untreated cases in 4-8 weeks.
- Complications:
 - <u>Acute</u>: internal hydrocephalus, cerebral infarction, cranial nerve palsies, convulsions, fluid & electrolyte disturbances.
 - <u>Chronic</u>: obstructive hydrocephalus, optic atrophy, subdural effusions spinal cord compression etc.
- ^o CSF shows rise in pressure, turbidity & a rise in lymphocytes upto 500- 1000 cells/cmm. A fine coagulum **(cobweb)** forms in the CSF when kept at room temperature for 6-24 hours; this is highly suggestive of tuberculous meningitis. Proteins are moderately increased. Sugar is reduced to less than 50 % of the blood sugar.

NON-TUBERCULOUS MYCOBACTERIA

- GROUP 1: Photochromogenic: produce yellow or orange pigment on exposure to light, example, M. kansasii, M. marinum.
- GROUP 2: Scotochromogenic: produce yellow, orange or reddish pigment in the *dark*, example, *M.scrofulaceum*.
- ^o **GROUP 3: Nonchromogenic:** does not produce pigment on exposure to light, example, *M. avium intracellulare, M. ulcerans*
- ^o GROUP 4: Rapidly growing mycobacteria: organism rapidly grows at 25^o C, example, M. fortuitum, M. chilonei.

Lesions caused by non-tuberculous mycobacteria

M. marinum (swimming pool bacillus, fish tank	Ulceration of the skin
bacillus)	
M. ulcerans	Extensive ulceration of the skin & subcutaneous
	tissue (Buruli ulcer)
M. kansasii	Pulmonary disease, local abscesses, bone & joint
M.scrofulaceum	lesion.
M. chilonei	Pulmonary disease, local abscesses,
M. avium intracellulare	Lymphadenopathy, pulmonary lesions, AIDS related

	& non AIDS related disseminated lesions.
M.scrofulaceum	lymphadenopathy

LEPROSY (Syn: Hansen's disease)

- Mycobacterium leprae. Portal of entry: abrasions in the skin & mucous membranes of upper respiratory tract.
- ° I.P: 2-7 years.
- $^\circ~$ In lepromatous leprosy males are twice more commonly affected than women.
- Cell mediated immunity (CMI) which develops against the lipid components of M. leprae determines the progress of disease.
- ^o Two polar forms are lepromatous & tuberculoid types.
- 0 It affects mainly peripheral nerves. It also affects skin, muscles, eye, bones, testes & internal organs.
- Leprosy is characterized by one of the following cardinal features:
 - Hypopigmented patches.
 - Partial or total loss of cutaneous sensation in the affected areas; the earliest sensation to be affected is **temperature**, specially the modality of cold sensation.
 - Presence of thickened nerves.
 - Presence of acid fast bacilli in skin & nasal smears.

Classification

- *i*. Indeterminate type: 1 or 2 hypopigmented macule. Bacteriologically negative.
- ii. Tuberculoid: one or 2 small well defined lesion. Bacteriologically negative.
- **iii.** Borderline: Bacteriological positivity of this lesion is variable. Satellite lesions are seen.
- **IV.** Lepromatous: diffuse infiltration, symmetrically distributed lesion. Collapse of nasal bridge, Gynaecomastia, anhidrosis.
- V. Pure neuritic: skin smear is negative.
- 0 In paucibacillary leprosy, the bacterial index is less than 2; in multibacillary leprosy, it is greater than 2.
- Cardinal signs of leprosy includes: loss of eyebrow, hypoanaesthic patches, ulnar nerve thickening.
- 0 Best diagnostic method for leprosy: split skin smear.
- Lepromin test: reaction is read at 48 hours (Fernandez reaction) & 21 days (Mitsuda reaction). If the diameter is more than 10mm at the end of 48 hours the test is positive. Lepromin test is **not a diagnostic test.** It is used in estimating the **prognosis.** The test is strongly positive in tuberculoid case. Lepromatous cases are Lepromin negastive.

CHLAMYDIAL INFECTIONS

PSITTACOSIS (Svn: Parrot fever)

- ° Chlamydia psittacii; primary pathogen of bird.
- 0 Chlamydia psittacii reaches the pulmonary alveoli & the reticuloendothelial cells of the spleen & the liver.
- ° I. P: 10 days.
- [°] Fever, sore throat, cough, pulmonary consolidation etc.
- ^o Respiratory failure & toxaemia are the usual causes of death (20%).

VIRAL INFECTIONS

- Only single stranded DNA virus
- Only double stranded RNA virus
- Naked DNA viruses are
- Maked RNA viruses are
- Segmented RNA viruses are Ŧ Ŧ
 - Circular viruses are dsDNA
- Parvovirus
- Reovirus
- Parvo, Adeno, Papova viruses
- Picorna, Calci, Reoviruses
- Bunya, Orthomyxo, Retro, Arenaviruses
- Papovavirus

Circular viruses are ssRNA Viral inclusion bodies

- Bunya, Arenavirus

Intracytoplasmic		Intranuclear	
Negri body	Rabies	Councilmann body	Yellow fever, Viral hepatitis
Guarnieri's body	Small pox	Cowdry type A	Herpes virus, Yellow fever
Henderson Paterson body	Molluscum		
	contagiosum	Cowdry type B	
Bollinger, Borrel's body	Fowl pox		Adeno virus, Poliovirus

Babes- Ernst, volutin granules: C. diphtheria
 Levinthal Cole Lillie bodies: Psittacosis.
 Miyagawa's granulocorpuscles: LGV.

VIRUS

Type of viruses	Names
DNA Viruses	
Adenovirus	Adenoviruses
	Herpes simplex virus types 1&2 (HSV)
	EBV
	Varicella-zoster virus
Parvovirus	Parvovirus B19
Pox virue	Vaccino virus
	Viral virus
Papovirus	Polyoma virus
RNA viruses	
Picornavirus	Poliovirus
	Coxsackie virus
	Rhinovirus
	Enterovirus
Togavirus	Rubella virus
	Alphavirus Flavivirus
Reovirus	Reovirus, Rotavirus
Orthomyxovirus	Influenza viruses A,B,C
Paramyxovirus	Measles virus
Rhahdovirus	Rahies virus
Arenavirus	Lassa virus
Retrovirus	HIV
Viruses associated with gastroenteritis	Rotavirus(groups A-E),
	Enteric adenovirus (type 40&41)
	Astrovirus
	Calcivirus
	Parovirus
Viral infections associated with a	Arbovirus infection
maculopapular rash	Adenovirus infection
	Enterovirus infection
	Measles
	Rubella, Rubeola
	Intectious mononucleosis Hopatitis B virus
Viral infections associated with haemorrhages	
Arbovirus, Mosquito borne	
	Yellow fever, Dengue haemorrhagic fever, Rift
	vallev fever, Chikungunya.

Tick borne	
	Kyassanur forest disease, Congo haemorrhagic fever.
Arena virus	
Paramyxovirus	Lassa fever, Epidemic haemorrhagic fever
Picornavirus	Atypical measles
	Acute haemorrhagic conjunctivitis

DISEASES CAUSED BY VIRUSES

Parvovirus B19	Frythema infectiosum (<i>fifth disease</i>): slapped face appearance.			
	Symmetric polyarthronathy in adults			
	Fetal abnormalities in utero			
	Anlastic crisis			
	Anaemia in patients with malignancy (eq. ALL)			
Human papilloma virus (HPV)				
Type 1	Plantar warts			
Type 3	Flat warts			
Type 5/8	Skin squamous cell carcinomas (SCC) in transplant patients			
Type 7	Warts in food handlers			
Type 6 & 11	Genital warts (condylomata acuminata): type 6 HPV.			
.,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,	Juvenile larvngeal papillomatosis			
Type 13	Oral leukoplakia			
Type 16	SCC cervix, penis, vulva.			
Type 30	SCC larvnx.			
Varicella / Zoster virus	Chickenpox, Herpes zoster.			
Herpes virus hominis				
(Herpes simplex)	Herpes labialis (Cold sore)			
Type 1	Keratoconjuctivitis			
	Whitlows			
	Primary stomatitis			
	Encephalitis			
	Genital infection (40%)			
	Genital infection (60%)			
Type 2	Neonatal infections			
Enteroviruses (1 & 2)				
 Coxsackie virus. 	Myocarditis, Pericarditis			
	Pharyngitis, Meningitis			
	Gastroenteritis			
	Bronholm disease			
	Neonatal infection			
2. Echovirus	Meningitis, Encephalitis, Conjunctivitis			
Lumphatuanhia vinuana (1.4)	Pharyngitis, Gastroenteritis, Neonatal Infection			
Lympnotropnic viruses (1-4)	Tufactious mononuelossia			
1. Epstein-Barr Virus (EBV)				
	Burkius lymphoma			
	Nasopharyngeal Carcinoma Haim Iaukoplakia (AIDS patiente)			
	Relative and atvaical lymphosysteric			
2 Cytomegalovirus (CMV)	Infection in immunocompromised natients-pneumonitis			
2. Cytomegalovirus (Chiv)	retinitis generalized infection Atypical lymphocytosis- less			
3 Human hernes virus	common			
6(HHV-6) (also called B-cell				
lymphotropic virus, HBLV)	Mononucleosis like syndrome in adults			
.,	Roseola infantum in children (exanthema subitum)			
	Atypical lymphocytosis			
	Nontender cervical lymphadenopathy			
4. Adenovirus				
	Acute pharyngitis in children, conjunctivitis+ Pharyngitis in			
	children			
	Acute follicular conjunctivitis in adults			
	Rarely pneumonia in adults			

HIV(Human immuno deficiency virus a retrovirus)		AIDS, infectious mononeucleosis like illness, Asymptomatic HIV infection, AIDS related complex (ARC also called prodormal AIDS), Persistent generalized lymphadenopathy (PGL) Thrombocytopenic purpura AIDS Dementia complex, Psychosis, Encephalitis
Influenza virus (A orthomyxo virus) Influenza A virus Influenza B virus Influenza C virus		Pandemics, epidemic of influenza Milder outbreaks in smaller groups e.g. school camps Rare

- The most common cause of sporadic encephalitis- HSV-1
- The most common cause of epidemic viral encephalitis- Arbovirus
- The most common cause of viral meningitis- Enterovirus
- Mumps virus does not cause pneumonia
- Measles virus does not cause Aseptic meningitis.

HUMAN HERPES VIRUS GROUP

Human Herpes virus	Common name
Туре I	Herpes simplex type-I
Type II	Herpes simplex type-II
Type III	Herpes Zoster virus type
Type IV	Epstein Barr type I
Type V	Cytomegalovirus
Type VI	Human B cell Lymphotropic virus
Type VII	R.K.virus

HUMAN IMMUNO DEFICIENCY VIRUS (HIV) INFECTION AND ACQUIRED IMMUNO DEFICIENCY SYNDROME (AIDS)

Causative organism: HIV virus; Lentivirinae sub-family of retroviruses; RNA viruses.

Two main types pathogenic to man are

- HIV-1:
 - Produce *more severe disease*.
 - Divided into group *M*(11 sub-types- *A* to *K*), Group O (9 sub types) and group N (new viruses)
 - C is the main sub-type of HIV 1 seen in India
- C is • HIV-2:
 - Producing milder disease.
 - Divided into 5 sub-types (A-E)
- * Pathogenesis:
 - ^o With the steady increase in the number of HIV, the *number of CD4+ lymphocytes* progressively fall. Normal CD4+ T cell count is 800-1200/ cmm.
 - ^o The *predominant opportunist infections* which affect the immuno-compromised host include:
 - Mycobacterium tuberculosis
 - Pneumocystis carinii
 - Fungi including candida, asphergillus, Cryptococcus, and histoplasma.
 - Protozoa including toxoplasma, Entamoeba histolytica and other amoebae, leishmania donovani, Giardia lamblia.
 - Helminthes such as Strongyloides stercoralis, scabies and possibly several others.
 - ^o *Infected monocytes carry the virus to the central nervous system* and lead to the development of lesions.
 - $^{\circ}\,$ The virus can be identified in the plasma by PCR and their number quantitatively determined.
 - Identification of HIV infection: Preliminary test to detect infection is the presence of antibody to HIV in serum by ELISA. Confirmation by immunoblot test (Western blot test).

ACQUIRED IMMUNE DEFICIENCY SYNDROME (AIDS)

- ♦ Majority of HIV infected persons develop AIDS with in 10-11 years after the infection.
- Rapid progressers develop disease within 2-3 years.
- Above 10% do not develop the disease due to protective genetic and HLA factors: non-progressers.
- **I.P:** Few months to 14 years (mean of 5.5 years

Clinical features:

- Initial symptoms include fever, headache, transient arthralgia, maculopapular rash, tender generalized lymphadenopathy, mouth ulcers, diarrhoea & rarely signs of encephalopathy.
- An early manifestation is persistent generalized lymphadenopathy (PGL).
- ✤ 3 groups:
 - ° **Group A**: Acute HIV infection & asymptomatic patients.
 - ^o **Group B**: symptomatic, but no AIDS specific features.
 - [°] **Group C**: those who have AIDS.
- CDC &WHO defined the following condition as suggesting AIDS in HIV positive individual:

Infections

- 1. Disseminated cytomegalovirus infection
- 2. Mucocutenous disseminated herpes simplex infection- exceeding 1 month duration.
- 3. *Infection by papovirus* giving rise to multifocal leucoencephalopathy.
- 4. *Tuberculosis* with CD4+ count <200/cmm- pulmonary or extrapulmonary.
- 5. Other mycobacteria: Kansasi, Avium intracellulare
- 6. Pneumocystis carinii pneumonia.
- 7. Candidiasis of oesophagus, bronchi and lung.
- 8. Cryptosporidiasis more than 1 month duration.
- 9. Toxoplasmosis of brain.
- 10. Isosporiasis.
- 11. Disseminated other *fungal infections-* histoplasmosis, Coccidioidomycosis, Cryptococcosis.
- 12. Extra intestinal strongyloidosis.
- 13. Lymphocytic interstitial pneumonia.

<u>Neoplasms</u>

- 1. Kaposi's sarcoma
- 2. Primary lymphoma- brain
- 3. Non-Hodgkin's lymphoma

Many infections which remain localized in immuno-competent subjects tend to become **generalized.** These include:

- **1.** Bacterial septicemias
- **2.** *M.tuberculosis*
- **3.** *M. avium intracellulare*
- 4. Toxoplasma gondii
- **5.** Cytomegalovirus and other viruses
- 6. Cryptococcus neoformans
- 7. Histoplasma capsulatum

COMMON VIRAL INFECTIONS OF THE RESPIRATORY TRACT

Disease	Causative agent	Clinical features	Complications	Diagnosis
---------	--------------------	-------------------	---------------	-----------

Coryza	Rhinoviruse s	 <i>I. P: 3-4 days</i> Headache, nasal congestion & obstruction. Mild fever & muscle pain. Lasts for 2-3 days. 	 Sinusitis Lower respiratory tract infection. Otitis media. 	Clinical diagnosis.
Influenza	3 groups of myxoviruse s Important among them: • Influenz a virus A • Influenz a virus B • Influenz a virus C	 I. P: few hours to 48 hours. Sudden onset with fever (38- 40°C), severe generalized myalgia, prostration &injected conjunctiva. Lasts for 3-4 days. 	 Pulmonary complications: Primary influenza virus pneumonia. Influenzal pneumonia with secondary bacterial infections. Bacterial pneumonia. Cardiac complications: Toxic myocarditis. Cardiac failure. Neurological complications: Febrile convulsion Meningitis Meningoencephalitis Encephalitis Reye's syndrome (hepatic failure with encephalopathy & rise in 	 Clinical diagnosis Isolation of virus from throat washings or sputum.
Parainfluen za	Parainfluen za viruses; paramyxovi rus group.	 I. P: 5-6 days Type 1: laryngotracheobronchitis or croup. Type 2: tracheobronchitis, bronchiolitis& bronchopneumonia 	 Secondary bacterial infection Otitis media Sinusitis Bronchiectasis 	 Sputum Clinical features.
Respirator y Syncytial Virus infection (RSV)	Paramyxovi rus family Pneumoviru s genus.	 Lower respiratory tract infection. <i>I.P: 3-5 days.</i> 	 Severe bronchiolitis leading to respiratory failure. Death in children below 5 years. 	 Demonstration of RSV antigens in nasal washings.

EXANTHEMES & ENANTHEMES

Disease	Causativ	Pathology	Clinical features	complications	Diagnosis
	e organism			• • • • • • • • • • • • • • • • • • • •	
Measles (<i>Rubeola</i> – red spots- Arabic)	organism Paramyxo virus (RNA virus)	 Droplet infection. Portal of entry: respiratory mucous membrane & conjunctiva. <i>Koplik's</i> spots in cheeks consists of vesicles & necrotic epithelium. Multinucleated giant cells with <i>Warthin</i> <i>Finkelday cells</i> (cytoplasmic inclusion bodies) found in the hyperplastic lymphoid tissues of lymph nodes, tonsils etc. 	 I. P: 9-11 days. High fever, excessive lachrymation, hacking cough & nasal discharge. Koplik's spots: 1-2 days before the rash. Red, maculopapular rash first appearing on forehead & behind the pinna & then spreading down to face, neck trunk & limbs. 	 Respiratory: a. Croup. b. Bronchiolitis. c. Bronchiolitis d. Pneumonia Eye:	 Leucopenia in early stages. Leucocytosis with secondary bacterial infection. CSF: raised protein & lymphocytosi s in encephalitis. Immunofluores cence: virus antigen can be detected. Multinucleated giant cells in Giemsa stained smears of nasal secretions.
Small pox (Variola)	<i>Variola virus</i>	Cytoplasmic inclusion bodies: <i>Guarnieri bodies</i> ; these are aggregates of the virus particles are called <i>Paschen</i> <i>bodies.</i>	 I. P: 12 days) Rash appears on 3rd or 4th day Rash is centrifugal; more on face & extremities. Axilla is usually free. Rash comes out as a single crop during 1-2 days. Rash is in the same stage all over. Multilocular & umblicated vesicles. Deep permanent scarring. 	 Secondary bacterial infection causes pneumonia, osteomyelitis, septicemia etc. CNS: Encephalitis Laryngeal oedema. 	 Clinical features Identificatio n of guarnieri bodies from the vesicular material. Isolation of the virus.

Chickenpo x (Varicella)	Varicella zoster virus (V- Z)	 Vesicles contain serum, polymorphs &multinucleated giant cells. Reye's syndrome may develop during acute phase especially in children and infants. 	 I. P: 14- 16 days. Skin rashes (exanthema) come <i>in crops</i> during the <i>first</i> <i>day of fever</i>. <i>Polymorphis</i> <i>m</i>: rashes of different stages may be present simultaneously . Rashes are <i>centripetal</i>. <i>Vesicles:</i> <i>unilocular</i>, <i>superficial</i>, <i>elliptical</i> & <i>clear fluid in</i> <i>the beginning</i> (<i>tear drop</i> <i>vesicles</i>). 	 Thrombocytopeni a →hemorrhage. Superadded staphylococcal infection. Bullous lesions in children with impetigo. Varicella gangrenosa: superinfection by hemolytic strerpyococci. Primary varicella pneumonia. CNS: meningitis, encephalitis. Congenital malformations: if it occurs during 1st & 2nd trimester of pregnancy. 	 Clinical diagnosis Tzanck smear: examination of material scraped from vesicles reveals intranuclear inclusion bodies &multinuclea ted giant cells. Isolation of virus. PCR: presence of virus in blood vessels can be detected.
Herpes Zoster (shingles)	Varicella zoster virus (V- Z) HSV	After chickenpox, V- Z virus becomes latent in ganglia along the entire neuraxis, particularly in trigeminal ganglia & the dorsal root ganglia, remaining mainly in the cytoplasm of the neurons. Attacks are precipitated by immunodeficiency states.	 Unbearable lancinating, deep-boring or burning pain. Sites of eruption: ophthalmic & maxillary divisions of trigeminal nerve, geniculate ganglion of facial nerve & thorasic & abdominal nerve roots. Rash may involve a whole dermatome or a part of it. The eruptions seldom cross the midline. Pain may persist for months or even years especially in elder patients: post- herpetic neuralgia. 	 Secondary infection. Ophthalmic herpes. Herpes of geniculate ganglion presents as facial nerve palsy & vesicles over ipsilateral auditory meatus (Ramsay -Hunt syndrome) Cervical herpes zoster →weakness of arm. Generalized herpes Congenital malformation of fetus in pregnancy. Post herpetic neuralgia. 	Clinical features.
Simplex (herpes	(Herpes simplex				

febrillis, fever blister, cold sore)	virus)/ Herpes virus hominis (HVH) HSV 1 HSV 2		 Recurrent crops of vesicles over the mucocutaneou s regions. Sexually transmitted 	 Herpes simplex encephalitis Kaposi's varicelliform eruptions (eczema herpeticum) 	• Virus can be isolated from the vesicle fluid.
Rubella (German measles- three days measles)	Togavirus ; RNA virus.	Lymph nodes are moderately enlarged (occipital, posterior auricular & posterior cervical lymph nodes), showing oedema & hyperplasia.	disease. I. P: 2-3 weeks. Congenital Rubella: fetal defects include retardation of growth, eye defects like cataract, glaucoma or retinopathy, heart lesions, deafness, mental retardation, hepatospleno megaly & skeletal abnormalities Compared to measles, constitution al symptoms are mild in children.	• Neurological complications rarely.	 Clinical features. Leucocyte count is normal in rubella.

<u>MUMPS</u>

- Mumps virus; group: paramyxoviruses.
- Infection spreads through droplets.
- Man is the only known host.
- I. P: 18 days (2- 3 week)
- Fever, Pain over the region of parotid gland, especially on opening the jaw, trismus, Sialoadenitis (usually bilateral) etc.
- Complications: in adult males (25%) orchitis, sterility if there is bilateral involvement, pancreatitis, meningitis (heralded by intense headache & meningeal irritation) etc.

CLINICAL AND EPIDEMIOLOGIC FEATURES OF VIRAL HEPATITIS

Features	HAV	HBV	HCV	HDV	HEV
Incubation (days) Agent	15-45 RNA virus	30-180 DNA virus seen as Dane particles under electron microscope.	15-100 RNA virus	30-180 RNA virus; which is incomplete & therefore it requires prior infection by HBV.	14-60 RNA virus transmitted mainly by the enteral route.
Transmission					

www.similima.com

Fecal-oral	+++				+++	
 Percutaneous 	Unusual	+++	+++	+++		
Perinatal		+++	+	+		
 Sexual 	+	++	+	++		
Clinical						
Severity	Mild	Occasionally	Moderate	Occasionally severe		Mild
-		severe		5-20%		
Fulminent	0.1%	0.1-1%	0.1%	Common		1.2%
 Progression to 	None	Occasional (1-	Common			None
chronicity		10%)	(50-70%)			
Carrier	None	1-30%	1.5-3%	Variable		None
Prognosis	Excellent	Worse with age	Moderate	Acute good, Chronic poo	or	Good

Serology of HBV

- * Hbs Ag: live virus infection; acute or chronic or carrier.
- Anti H bs Ag: *immune*, cure & no active disease.
- Anti Hbc Ag:
 - Ig M Anti Hbc Ag: new infection.
 - Ig G Anti Hbc Ag: old infection.
- * Hbe Ag: high infectivity
- Anti Hbe Ag: low infectivity.

ENTEROVIRUSES

POLIOMYELITIS (syn: Heine Medin Disease, Infantile paralysis)

- Polio virus: Picorna viruses (group).
 - Three types: type1, 2 & 3; Type 1 is most common & responsible for the majority of paralytic cases.
- Disease is spread by the *feco- oral route*.
- In paralytic cases, anterior horn cells of the spinal cord & the medullary nuclei are swollen & congested. Classic paralytic polio develops only rarely.
- In the early stages, the CSF shows increase in cells, mainly polymorphs but this gives way to lymphocytic increase with the passage of time. Protein level: moderately increased. Sugar level: normal.
- * I. P: 7- 14 days.
- Clinical features: initial symptoms are headache, fever, muscle pain & diarrhea.
 - Nonparalytic polio: features of meningitis.
 - **Paralytic polio:** paralysis may involve *spinal group of muscles, bulbar muscles* or a combination of both.
 - Spinal form: predilection for the cervical & lumbar segments of the spinal cord. Paralysis is asymmetrical. If there is total destruction of neurons complete paralysis occurs.
 - **Bulbar form:** affection of *cranial nerves* occurs early in this form *dysphagia, dysarthria* & *dysphonia* occurs commonly. Facial palsy develops less commonly.
 - **Polioencephalitis:** alteration in the level of consciousness, convulsions, signs of brainstem involvement & varying combinations of spastic or flaccid paralysis.
- Post-polio syndrome (PPS): Progressive muscular atrophy & muscle weakness occurring several years after apparent recovery from poliomyelitis with residual paralysis. CSF shows elevation of IgM & interleukin-2 which point to an immunological basis for this syndrome.
- * Prevention:
 - Live vaccine: Sabin (given orally)
 - Killed vaccine: Salk vaccine (subcutaneously)

Regime for immunization (recommended by IMA)

Contact	Age of child	Vaccine	Other care
1	At birth	BCG, OPV	
2	6 weeks	DPT, OPV	
3	10 weeks	DPT, OPV	

4	14 weeks	DPT, OPV	
5	9 months	Measles	Vitamin A
6	16 months (booster)	DPT, OPV	Vitamin A

ECHOVIRUSES (Syn: Enteric cytopathogenic human orphan viruses):

RNA viruses which produce a spectrum of diseases ranging from mild upper respiratory tract infection, fever with rash to Acute Hemorrhagic Conjunctivitis (AHC).

- Acute Hemorrhagic Conjunctivitis (AHC):
 - Spinal form
 - Cranial form
 - Combined form

COXSACKIE VIRUS INFECTION

- Picorna viruses A &B.
- Syndrome of pleurodynia (devils grip, Borholm's disease, epidemic myalgia) is caused by coxsackie B group viruses. Severe pain develops in chest & upper abdomen after a prodromal febrile illness.
- Hand –Foot & Mouth disease: caused by Coxsackie viruses types A-4, 5, 7,9,10 & 16. Febrile illness followed by superficial vesicular eruptions inside the mouth, fingers, palms & soles.

RABIES (Hydrophobia, Lyssa)

- > RNA viruses; *rhabdoviruses*.
 - *Street viruses:* when isolated first from animals. It has long incubation periods & it can multiply in salivary glands.
 - *Fixed viruses:* it has shorter incubation periods & it can't multiply in salivary glands.
- > Intracytoplasmic inclusion bodies: **Negri bodies.**

> Clinical features:

- I. P: 10 days to more than 2 months.
- Neurological involvement leads to *spatic & paralytic form.*
 - **Spstic form:** Hydrophobia confirms the diagnosis (though not pathognomonic). Death can occur due to *respiratory paralysis* resulting from *bulbar involvement*. Whole course extends to less than 5 days.
 - **Paralytic form:** ascending paralysis. This occurs more often after vampire bat bites.
- Neutralizing antibodies against rabies virus can be demonstrated after the initial 10 days of onset.
- > Virus can be isolated in saliva or CSF for the initial 2 weeks of illness.
- Active immunization:
 - Antirabies vaccination (ARV) introduced by Louis Pasteur. All vaccines are killed vaccines.
 - Avian embryo vaccine: these are produced in chick (Flury) or duck embryo. Neuroparalytic accidents are less common with this vaccine. Used for active immunization of dogs & pets.
- Cell culture vaccines:
 - Monkey kidney cell vaccine
 - Purified chick embryo cell vaccine
 - Human diploid cell vaccine (HDCV)
- > Passive immunization: Human rabies immunoglobulin HRIG. If the risk is class 3 or even 2, passive immunization should be given.
- Pre-exposure immunization: 3 doses of cell culture vaccines on day 0, 7, 21 or 28 days; for 2 years.

Classification of risk

Class1 (light risk)	Class 2 (moderate risk)	Class 3 (great risk)
Licks except on face & fingers	Licks on fresh wounds & cuts & abrasions on fingers	Licks on fresh cuts, scratches & bits on head, neck or face.

Licks on intact mucous membrane		Bites on fingers & all lacerated wounds
Light bits, 9 sevetable, ever parts of the body	Scratches c	n
except head neck face & fingers	fingers	lackal & wolf bites
Consumption of unboiled milk or handling raw		Any class 2 patient who has not
flesh of rabid animal		received RV within 2 weeks

ARBOVIRUSES

1. DENGUE FEVER (BREAK BONE FEVER)

- Of all the arthropod- borne viral diseases, Dengue fever is the *most common*.
- Group B arbovirus.
- Main vector is Aedes aegypti mosquito.
- * Clinical features:
 - *3 types*:
 - ^o Classical Dengue Fever
 - ^o Dengue Haemorrhagic fever without shock
 - ^o Dengue Haemorrhagic fever with shock
 - Incubation period 3 to 10 days.
 - The onset is sudden with chills and high fever, intense headache, muscle and joint or bone pains which prevent all movement.
 - Mottling, or fleeting pin point eruptions on face, neck, chest and limbs *sparing palms and soles* during the first half of the febrile period and a conspicuous rash that may be maculopapular or scarlatiform on 3rd or 4th day.
 - The fever may rise again producing a *saddle-back fever curve*.
 - Dengue haemorrhagic fever is confined exclusively to children less than 15 yrs of age. There may be plasma leakage and abnormal haemostasis, as manifested by a rising haematocrit value and moderate to marked thrombocytopenia.
 - In dengue shock syndrome shock is present along with all the above criteria.

SANDFLY FEVER (Syn: phlebotomous fever, Papatasii fever, 3 days fever)

- Transmitted by the sandfly *phlebotomous papatasii (female)*
- I.P: 2-6 Days
- Absence of rash & lymphadenopathy helps to differentiate it from dengue fever.
- Leucopenia.

YELLOW FEVER

- Group B arbovirus.
- Transmitted by aedes mosquitoes
- ✤ I.P: 3-6 Days
- It is a zoonotic disease affecting principally *monkeys and other vertebrates*.
- It shares clinical features of *dengue fever* but is characterized by more severe *hepatic and renal* involvement.
- 3 stages: first stage; most of death occur at this stage; second stage; third stage: intoxication follows within hours to days in severe cases.
- ✤ Vaccination : live attenuated vaccine (17D strain)

CHIKUNGUNYA (Chicken Guinea)

- Form of viral fever *resembling dengue fever*
- Chikungunya virus; Alphavirus (group A arbovirus).
- Transmitted by Aedes aegypti mosquito, though recent research by the Pasteur Institute in Paris claims the virus has suffered a mutation that enables it to be transmitted by Aedes Albopictus (Tiger mosquito).
- The name is derived from the word Makonde meaning "that which bends up" in reference to the stooped posture developed as a result of the arthritic symptoms of the disease
- * Clinical features:

- ^o Incubation period 3-12 days.
- ² Sudden onset of *flu-like* symptoms including a severe headache, chills, fever (>40°C, 104°F), joint pain (especially on the 4th day), backache, nausea, vomiting, *petechial or maculopapular rash usually involving the limbs and trunks. Migratory polyarthritis mainly affects the small joints* of the hands, wrists, ankles and feet with lesser involvement of the larger joints. Joints of the extremities in particular become swollen and painful to touch.
- ^o The disease has a *biphasic* course also. Following 1-6 days of fever, the temperature returns to normal for 1-3 days and then there is a second period of fever for a few days.

JAPANESE ENCEPHALITIS (JE)

- Group B arbovirus.
- Virus has been isolated from *culex mosquitoes, anopheles mosquitoes and mansonia.*
- Clinical features: The disease has 3 phases:
 - ° Prodromal
 - ^o Acute encephalitic stage
 - [°] Convalescence (recovery phase)
 - $^{\circ}\,$ There may be fever, headache, and altered sensorium including coma, convulsion, and neck rigidity.

KYASANUR FOREST DISEASE (KFD)

- ✤ Group B arbovirus.
- Vectors: tiks; Hemaphysalis spinigera &Ixodes.
- Man gets the infection by the bite of the nymph of *H. spinigera*
- I. P: 2-7 days.
- Febrile illness characterized by fever with rigor, meningism, delirium, mental confusion etc.

SYSTEMIC FUNGAL INFECTIONS

- Candidiasis: Candida albicans; in genitalia it usually presents as vulvovaginitis in diabetic women.
- Aspergillosis: A. fumigatus is most common. Healed tuberculous cavities or other cavities may be the seat of colonization of the fungus.
- Histoplasmosis (Darling's disease or cave disease): Histoplasma capsulatum. Pulmonary lesions may resemble different types of tuberculosis.
- Cryptococcus (Syn: Torulosis, European Blastomycosis, Busse- Bushke's disease): Cryptococcus neoformans; often the lesions are mistaken for tuberculosis.
- Rhinosporidiosis: Rhinosporidium seeberi; affects the mucous membrane of the nose, larynx, eyes, ears, mouth, genitalia, rectum & skin.

ACTINOMYCES & NOCARDIA.

ACTINOMYCOSIS (Ray-fungus disease)

- Actinomycetes are bacteria. They form mycelia like fungi.
- Actinomyces Israeli.
- Disease present as:
 - Cervicofacial form (extensive destructive lesions in the presence of minimal general symptoms).
 - Thorasic form (lesion resemble TB)
 - Abdominal form(disease may present as acute appendicitis)
 - Bony lesions (osteomyelitis)

MYCETOMA (Madura foot, Maduramycosis)

- Chronic granulomatous inflammation caused by several species of actinoimycetes & fungi.
- Nocardia madurae, N. asteroids & N. brasiliensis.

PROTOZOAL DISEASES

<u>MALARIA</u>

Agent: protozoa of the genus plasmodium.

- Airport malaria: occurrence of malaria in people living near airports or working in airports, caused by bites of infective mosquitoes arriving in aircrafts.
- Vectors: female anophelene mosquitoes.
- *The cycle: Sexual cycle (sporogony) in mosquitoes & schizogony (asexual cycle) in man.*

Characteristic features of human malarial parasite

Colour of pigment

1. Plasmodium vivax		-	yellow brown
2. P.ovale		-	dark brown
3. P. malaria		-	brown black
4. P.falciparum	-	black	

Number of merozoites per schizonts (trophozoite divides by binary fission to form schizont, which contains several merozoites).

1. Plasmodium vivax		-	8-16
2. P.ovale		-	8-12
3. P. malaria		-	6-12
4. P.falciparum	-	12-24	

Features P.vivax(benign tertian malaria)		P.malariae(quartan malaria)	P.falciparum (malignant tertian)	P.ovale (ovale malaria)
Incubation period	14 days	28 days (longest)	12 days (shortest)	17 days
Duration of erythrocytic cycle(schizogony)	48 hours	72 hours	48 hours	48 hours
Red cell preference	Reticulocytes	Older cells	Younger cells	Reticulocytes
Exoerythrocytic cycle of relapse	Present	Absent	Absent	Present
Changes seen in infected RBC	Enlarged	Not enlarged	Not enlarged	Slightly enlarged
Dots	Schuffner's dots	Ziemann's dots	Maurers clefts	Schuffner's dots
Gametocytes	Round or oval	Round or oval	Crescentic banana shape	Round or oval
Trophozoites	Amoeboid	Band shape	Compact	Compact
Forms in peripheral blood	All stages are present		Gametocytes and ring form of trophozoites	
Hypnozoites (dormant sporozoite)	present	Absent	Absent	Present

Clinical features of malaria

Vivax malaria	Ovale tertian malaria	Falciparum malaria	Malariae malaria (quartan malaria)
Paroxysms in 3 stages: · Cold stage · Hot stage · Sweat stage	Similar to Vivax malaria.	Prodromal symptoms are severe than vivax malariae.	
Paroxysm repeats at interval of 48 hours.		Periodicity may be either <i>quotidian (daily) or sub tertian</i> (less than 48 hours)	Fever occurs with <i>quartan (every 72 hours)</i> periodicity.
Patient feel well in between the febrile paroxysms		Patient may not feel well in between the febrile paroxysms.	It may take several weeks to develop periodicity.

Severe falciparum malaria (any one of the following clinical presentation)

- 1. cerebral malaria
- 2. Severe anaemia PCV <15%, Hb <5g/dL, parasitemia>10000/ μL.
- 3. Renal failure urine output 480mL/ 24 hours in adults or 12mL/kg/24 hours in children & creatinine >3mg/dL
- 4. Pulmonary oedema or respiratory distress syndrome
- 5. Hypoglycemia; whole blood glucose <40 mg/dL
- 6. Shock
- 7. Spontaneous haemorrhage
- 8. Repeated generalized seizures
- 9. Acidosis
- 10. Haemoglobinuria.
 - Algid malaria: this occurs in patients with predominantly gastrointestinal manifestations. They become extremely dehydrated &shocked without any warning signal.
 - Black water fever (BWF): sudden intravascular hemolysis gives rise to fever, rigor, anemia & hemoglobinuria. If severe shock, jaundice & hemoglobinuria supervene.

Diagnosis

- *Blood smears* are taken at the *onset of paroxysm.* The general characteristics of plasmodia in Romonowsky- stained films are:
 - [°] Location within erythrocytes
 - ° Red chromatin
 - ° Blue cytoplasm.
- Fluorochrome stains & quantitative buff coat (QBC) analysis.
- Fluorescence activated cell sorter (FACS) can detect paralyzed erythrocytes.
- Antigen detection tests like ELISA.
- PCR.

LEISHMANIASIS:

Pathogenic Leishmania are

- 7 Leishmania donovani causing Kala-azar (visceral leishmaniasis) and Post-Kala-azar dermal Leishmaniasis.
- 7 L.tropica and L.major causing **Oriental sore- cutaneous form.**
- 7 Several sub species of L.braziliensis and L.mexicana causing mucocutaneous Leishmaniasis.

KALA-AZAR: (Visceral Leishmaniasis)

- The name Kala-azar (black *fever*) is derived from the dark pigmentation that develops in the victims of visceral Leishmaniasis.
- *Males* are affected more than females.
- Main vector sand fly- Phlebotomus argentipes (females).
- Kupffer cells are distended with LD (Leishman-Donovan) bodies.
- Cell-mediated immunity is impaired.
- I.P: 2-6 months
- Periods of pyrexia and apyrexia alternate for several months.
- Occasional patient may show two peaks of fever in 24 hours. *This double rise is very suggestive of Kala-azar.*
- Despite the fever *patient feels well and has good appetite.*
- Progressive emaciation, anaemia and hepatosplenomegaly.
- **Napier's aldehyde test** is a simple bedside test helpful in the *diagnosis of chronic kala azar*.
- Skin test- Leishmanin test of Montenegro has been used to elicit delayed hypersensitivity. Test is negative in active kala azar. The test is positive in cutaneous leishmaniasis.

POST-KALA-AZAR DERMAL LEISHMANIASIS (Syn: PKDL- Dermal Leishmanoid of Brahmachari)

- Chronic granuloma of the skin caused by Leishmania donovani following recovery from kala azar.
- * Clinical features: Hypopigmented macules & nodular lesions.
- Aldehyde test is usually negative.

CUTANEOUS LEISHMANIASIS (Syn: Oriental sore, Delhi boil, Baghdad boil, Chiclero's ulcer)

- Primary infection of the skin caused by direct inoculation of the parasites at the site of lesions by the appropriate vector sandflies.
- Vector: female sandflies; P. papatasii.

AMERICAN CUTANEOUS & MUCOCUTANEOUS LEISHMANIASIS (Syn: Espundiasis)

- Cutaneous lesion resermble s oriental sore.
- Mucocutaneous form (espundia): the initial ulcer is followed by direct extension or metastatic spread from a distant lesion to the mucosa of nose & pharynx.

TRYPANOSOMIASIS

AFRICAN TRYPANOSOMIASIS (Syn: Sleeping sickness)

- Cause by 2 subspecies of *Trypanosoma Brucei*, *T. gambiense* (*Gambien sleeping sickness*) & *T. rhodesiense* (*Rhodesian sleeping sickness*).
- Transmitted by *tsetse flies (Glossina)*.
- * I. P: 1-3 weeks.
- * The initial lesion is the formation of a nodule at the site of bite: trypanosomal chancre.
- Lymph nodes are more pronounced in the posterior triangle of neck in **T. gambiense** infection. Enlargement of these groups of lymph nodes are called **Winterbottom's sign.**
- Unusual manifestations: circinate erythematous skin rashes, localized oedema over face, eyelids & neck, ulnar hyperaesthesia (kernadel's sign) neuralgic pains, myocarditis, pericardial effusion, jaundice etc.
- CNS symptoms are more marked in T. B. gambiense.

AMERICAN TRYPANOSOMOASIS (Syn: Chagas Disease)

- Trypanosoma cruzi (schizo-trypanum).
- Disease is transmitted by Reduvid bugs.
- Infected mothers can transmit the disease to the fetus, giving rise to congenital Chagas disease.
- In children disease occurs in an acute form presenting with chagoma, generalized lymhadenpoathy, fever, myocarditis etc.
- Initial lesion presenting with unilateral oedema of the conjunctiva & adjuscent tissues is called Romana's sign.

OTHER PROTOZOAL INFECTIONS

AMEBIASIS

- Entameba histolytica; it exists in 2 forms- the cyst & trophozoite.
- * Clinical manifestation:

> Intestinal amoebiasis

- Acute amoebic dysentery
- **Non dysenteric amoebiasis:** an amoebic granuloma (*amoeboma*) may be felt as a sausage shaped mass in the right iliac fossa.
- Extra-intestinal amoebiasis:
 - *Hepatic amoebiasis:* almost all patients give a history of alcoholism.

PRIMARY AMEBIC MENINGOENCEPHALITIS

- Naegleria gruberi; free living soil amebae. Hartmanella (Syn: Acanthamoeba) can also affect man.
- Organisms enter through the roof of nose &spread up the cribriform plate to reach the subarachnoid space.

GIARDIASIS

- Giardia intestinalis (G. Lamblia)
- Presenting symptoms range from mild abdominal discomfort to explosive diarrhea.

BALANTIDIASIS

- Balantidium coli; large ciliate.
- These resemble mild to moderately severe amoebic dysentery.

TOXOPLASMOSIS

- Toxoplasma gondii.
- Full life cycle in cats & other canine hosts.
- Congenital toxoplasmosis: neurological involvement.
- Hepatosplenomegaly, thrombocytopenic purpura, lymphadenopathy & rashes have been described.
- * **Toxoplasmin skin test:** intradermal test using toxoplasma antigen.

CRYPTOSPORIDIOSIS

- Cryptosporidium parvum.
- Acute watery diarrhea with fever & malaise.
- Cryptosporidiosis is a common opportunistic infection in AIDS.

INTESTINAL NEMATODES

ASCARIASIS

- Ascaris lumbricoides.
- During the stage of larval migration, pulmonary symptoms like cough, wheezing & hemoptysis may occur: Loeffler's syndrome; common cause of respiratory symptoms in children.

HOOKWORM INFECTION (Ancylostomiasis)

- Ancylostoma duodenale & Necator americanus.
- During the stage of larval migration, allergic symptoms like cough, dyspnoea, osinophilia & hemoptysis may occur: Loeffler's syndrome.

TRICHURIASIS (Whipworm infection)

Trichuris trichuria.

STRONGYLOIDIASIS

- Strongyloides stercoralis.
- Malabsorption syndrome or chronic diarrhea resembling Crohn's disease.

ENTEROBIASIS (Oxyuriasis, Thread worm, Pinworm, Seatworm)

Enterobius vermicularis (Oxyuris vermicularis)

TRICHINOSIS (Trichiniasis, Trichnellosis)

- * Trichinella spiralis; parasite of several animals like pig, rat, polar bear etc.
- * Intercostals muscles, pectoral muscles, diaphragm & shoulder girdle muscles are heavily affected.
- Eosinophilia is the constant finding in early stages.

LARVA MIGRANS

- Visceral Larva Migrans: manifestation produced by larvae of non-human ascarides when they migrate through the human tissues. Most often ascarides of dogs & cats- Toxocara canis & Toxocara catis. It is self limiting, since the encysted larvae die out after varying periods.
- Cutaneous Larva Migrans: larvae of Ancylostoma braziliense & A. canicum (dog's & cat's hookworms) & Strongyloides stercoralis cause creeping eruption.

CESTODIASIS (Syn: Tapeworm infection)

TAENIASIS SAGINATA (Syn: Beef Tapeworm, Unarmed Tapeworm)

- Taenia saginata; commonest among large tapeworm found in man.
- ✤ Man is the definitive host.
- Cystic stage: Cysticercus bovis.
- ✤ T. saginata segment has more than 15 lateral branches.

TAENIASIS SOLIUM (Syn: Pork Tapeworm, Armed Tapeworm

- Taenia solium; adult inhabits the small intestine of man.
- The name solium is derived from the shape of the rostellum which resembles the conventional figure of the sun.
- Man is the definitive host & pig is the intermediate host.
- Cystic stage: Cysticercus cellulosae (bladder worm).
- Heavily infected pork: *measly pork.*

ECHINOCOCCOSIS (Syn: Dog Tapeworm, Hydatid Worm)

- ♦ Larval form of Echinococcus granulosus; less commonly E. multilocularis.
- Definitive hosts are dogs, wolves, jackals & other canines.
- Hydatid thrill: place 3 fingers of left hand firmly over enlarged liver & percuss on the middle finger with the right hand & feel the sensation over the other 2 fingers, produced by the free floating daughter cysts in the cyst cavity
- * **Casoni's reaction:** this test is suggestive but not specific.

DIPHYLLOBOTHRIASIS LATUM (Syn: Dibothriocephalus latum)

• Two intermediate hosts are required: *fresh water cylops and fresh water fish.*

HYMENOLEPIS NANA (Syn: Dwarf Tapeworm)

Smallest tapeworm found in man

HYMENOLEPIS DIMENUTA

- Parasite primarily affecting rats and mice.
 DIPYLIDIUM CANINUM
 - Tapeworm seen in cats and dogs

TREMATODE (FLUKE) INFECTIONS

FASCIOLIASIS (Syn: Sheep Liver fluke)

- Fasciola hepatica.
- Snail vector: L; imnaea truncatula. The cercariae encyst on aquatic vegetation
- ✤ Man gets the infection by eating contaminated watercress.

HETEROPHYIASIS

- **Heterophyes:** a small fluke found in small intestine of man, dog, cat & wolf.
- They resemble Clonorchis sinensis.
- Snail vector; Pironella conica.

CLONORCHIASIS (Syn: Chinese liver fluke)

- Infection of the *biliary passages* by *clonorchis sinensis* causes clonorchiasis.
- In India it has been reported among the Chinese immigrants.

SCHISTOSOMIASIS (Syn: Bilharziasis)

- Infection by Schistosoma haematobium, S. mansoni (both parasitize only man) or S. japoniocum (affect man & other animals)
- Schistosoma haematobium reaches the bladder & other pelvic organs. The eggs appear in urine.

- Schistosoma japonicum affects the small intestine & the ascending colon.
- Schistosoma mansoni lodges in the descending colon & rectum.
- The eggs of Schistosoma japonicum & Schistosoma mansoni are passed in feces.
- Clinical features:
 - Swimmer's itch or cercarial dermatitis: itching & popular rashes at the site of penetration.
 - **Katayama syndrome**: marked *eosinophilia* develops in about 4-6 weeks (2nd stage).
 - Late complications:
 - Schistosoma haematobium (Genitourinary schistosomiasis)
 - Schistosoma mansoni (intestinal schistosomiasis)
 - Schistosoma japonicum (Katayama disease, Asciatic schistosomiasis): its pathogenicity is greater since it produce more eggs.

TISSUE NEMATODES

LYMPHATIC FILARIASIS

- Caused by **Wuchereria bancrofti & Brugia malayi.**
- Adult worms living in the lymphatic vessels are responsible for the lesion. Males are smaller than females.
- Microfilaria (embryonated eggs) is numerous in the peripheral blood at night.
- Vectors: many species of Culex & some species of Anopheles mosquitoes.
- Commonest clinical presentation is lymphoedema involving the extremities or genitalia & at the other end of the clinical spectrum tropical pulmonary oesinophilia (TPE) syndrome.
- * Spectrum of disease in lymphatic filariasis:
 - ° Endemic normals
 - [°] Asymptomatic microfilaraemia
 - ° Clinical disease
 - [°] TPE syndrome.
 - ICT (Immunochromatographic test: both card & ELISA based ICT are highly specific & sensitive for the diagnosis.

Manifestations of lymphatic filariasis

Early silent phase	Clinically asymptomatic. Microfilaria present peripheral blood. Dilatation of lymphatics demonstrated by ultrasonography & lymhoscintigraphy.
Acute manifestations	Acute adenolymphangitis Acute epididymo-orchitis, funiculitis Acute onset hydrocele due to inflammation Abscess formation Acute abdominal lymphadenitis Haematuria
Chronic manifestations	<i>Lymphoedema/ elephantiasis of extremities, genitalia & breasts</i> <i>Hydrocele</i> <i>Lymph scrotum</i> <i>Chyluria (sometimes pyuria with disintegration of the cells may appear milky:</i> <i>pseudochylous urine) , chylocele, chylous ascitis.</i> <i>Lymph node enlargement</i> <i>Lymphadeno-varix.</i>

LOIASIS

- Loa loa; adult worm migrate in the subcutaneous tissues of various parts including the eyes.
- Infection is transmitted by Chrysops (C. silacea & C. dimidiata).
- Disease manifests as Calabar swellings which are recurrent localized allergic inflammatory swellings.
- Diagnosis confirmed by demonstration of microfilaria in peripheral blood during day time.
 ONCHOCERCIASIS (Syn: African river blindness)
 - Cutaneous form of filariasis.
 - Onchocerca volvulus.

- ✤ Vectors: female black flies; genus simulium.
- Firm, freely movable subcutaneous nodules commonly seen around head & shoulders or the pelvic girdle.
- Intense itchy lesions. Later skin becomes thickened & hyperkeratotic (Crocodile skin).
 Ocular lesions ultimately lead to blindness.

OTHER FILARIAL INFECTIONS

- Dipetalonema perstans: adults are seen in subserosal layer of viscera. Vectors: blood sucking ** insects-culicoides.
- ٠ Dipetalonema streptocerca: Vectors: blood sucking insects-culicoides. Adult worms are seen in dermis & subcutaneous tissues.
- * Mansonella ozzzardi: adult worms are found in the visceral adipose tissue. Vectors include simulium & culicoides.

DRACONTIASIS (Syn: Guinea worm, Serpent worm, Dragon worm)

- Dracunculus medinensis.
- Intermediate host: fresh water Cyclops.
- Urticaria, eosinophilia, nausea, vomiting, diarrhea etc.
- Ulcer may develop & it may act as a portal of entry for clostridium tetani.

RARE HELMINTHIC INFESTATIONS

- Multiceps Multiceps: this is a tapeworm, adult of which live in the intestines of dog. Larvae (bladder worm) are seen in the brain of sheep & other herbivores- Coenurus cerebralis.
- **Intestinal capillariasis:** the adults (*Capillaria philippinensis*) parasitize birds.
- * Anisakiasis: this is caused by Anisakis species which are intestinal nematodes of marine animals.
- * Angiostrongyliasis: infection by Angiostrongylus costaricensis or Angiostrongylus cantonensis. Man gets infected by eating molluscus, prawns or crabs which harbors the larvae.
- * Gnathostoma spinigerum: intestinal nematode afeecting dogs & cats.
- * Sparganosis: Sparganum is the second larval stage of tapeworm of the genera Spirometra & Diphyllobothria. Ova are ingested by Cyclops & the 1st stage larvae (procercoids) develop in them.

CLASSICAL PRESENTATIONS OF PARASITIC DISEASES

Presentation	Parasitic Disease
"Anchovy sauce" expectoration	Amoebic abscess
"Grape skin" expectoration	Hydatid disease
Duodenal ulcer type pain	Hookworm, Strongyloides stercoralis
Intestinal obstruction	Round worm
Rectal prolapse	Whip worm, Trichostrongylus
Pruritus ani	Pinworm, Thread worm
Lymphoedema, elephantiasis	Filariasis
Pulmonary hypertension	Schistostoma mansoni
Portal hypertension	Schistosoma Japonicum
Terminal haematuria	Schistosoma haematobium
Haemoptysis	Paragonomiasis
Cholangitis, pancreatitis	Clonorchis sinensis
Cholangiocarcinoma	Chagas disease
Megaoesophagus/-colon	Giardiasis
Cardiomyopathy	D.latum
Malabsorption	Hookworm
Vit. B12 deficiency	
Iron-deficiency anaemia	

DISEASES CAUSING AGENTS

DISEASE	AGENT
Rubella	Rubella virus (a togavirus)
Mumps	Mumps virus (a paramyxovirus)
Yellow fever	A togavirus
Dengue fever	A togavirus
Trachoma	Chlamydia trachomatis

Q-fever	Coxilla burnetti (Rickettsia like organism)
Diphtheria	Corynebacterium diphtheriae
Anthrax	Bacillus anthracis
Bacillary dysentery	Shigella dysenteriae, flexneri, sonnei
Tularaemia	Francisella tularensis (transmitted by flies
	and ticks)
Oroya fever	Bartonella baciliformis (transmitted by
	sand flies)
Brucellosis (undulant fever)	Brucella melitensis
Gianders	Pseudomonas mallel
Syphilis Concerning and the limits account or with	Neisseria generrheese
Gonorrhoed, ophthalmia neonatorum	Columnatobactorium granulamatia
	Chlamydia trachomatic (l 1 2 3 coro
	types)
Nonspecific urethritis, proctitis, cervicitis	Chlamydia trachomatis
	Mycoplasma hominis
	Ureaplasma Urealvticum
	Trichomonas vaginalis
	HSV
Acute pelvic inflammatory disease	N.gonorrhea
	Clamydia trachomatis
AIDS	HIV 1 & 2
Molluscum contagiosum	Pox-like virus
Genital warts	Pailloma virus
Hepatitis	HBV,HCV, CMV
Vaginal thrush, balanitis	Candida albicans
Vaginitis, urethritis, balanoposthitis, (protozoal)	Trichomonas vaginalis
Genital scabies	Sarcoptes scabei
Pediculosis pubis	Phthirus pubis
Syphilis	Treponema pallidum
Bejel	Treponema pallidum variant
Pinta	
Taws Canicala favor	
	Leptospira canicola
Louso-borno rolansing fovor	Borrolia recurrontic
Tick-borno rolansing fovor	Borrelia dutonii
Cancrum oris	Borreli vincenti
Lyme disease	Borrelia burgdorferi
Psittacosis (ornithosis, a zoonosis)	C psittaci
Kala-azar	L donovani (transmitted by female sand
	flies)
Espundia (mucocutaneous leishmaniasis)	L.braziliensis
Diffuse cutaneous leishmaniasis	L.amazonensis
Sleeping sickness	T.brucei (bite of tsetse fly of either sex)
Chagas disease	T.cruzi (transmitted by faeces of a reduvid
	bug)
Toxoplasmosis	Toxoplasma gondii
Tertian malaria	P.vivax and P.ovale
Quartan malaria	P.malariae
Malignant tertian malaria	
Cerebral malaria	
Algid malaria	P.falciparum
Septicaemic maiaria	
Didukwaler lever	Echino cocoura area ula sus
nyualiu disease	Echnococcus granulosus and
scabios	Sarcontos scaboi (a mito)
Tinea versicolor	Malassazia furfur
Meningitis 2months to 2 years	
Meningitis after 12 years	Meningococcus
	Corvnehacterium acnes
	Corynebacterium aches

Sodoku	Spirillum minus
Favus	Trichophyton schonleinei
Fungus ball in lung cavity (aspergillosis)	Aspergillus fumigatus
Multiple cavities in lung (blastomycosis)	Blastomyces dermatitidis
Sinusitis	Pneumococcus

ARTHROPOD BORNE DISEASES

Name of arthropod		Diseases	
House fly,	Musca	Dysentries, Salmonelloses, Enteric fevers, Cholera	
	Tebanide	Tularaemia, Anthrax	
Tsetse fly,	Glossina	African trepanosomiasis	
Black fly,	Simulium	Onchocerciasis	
Sand fly,	Phlebotomus	Leishmaniasis, Bartonellosis	
Mosquito,	Anopheles	Malaria, some arboviruses	
	Culex	Bancroftian and Brugian filariasis	
	Aedes	Yellow fever, dengue, Chickangunya, other arboviruses	
Soft tick, Hard tick, Fleas, Mites, Lice,	<i>Ornithodoros Ixodidae Xenopsylla Leptotrombidium Allodermanysus Pediculus</i>	Lyme disease, Tick-borne relapsing fever Some typhus fevers, Kyasanur forest disease,Tularaemia Endemic typhus, Plague Scrub typhus Rickettsial pox Epidemic typhus, louse-borne relapsing fever, trench fever, Diphylidum caninum	
Winged bug, Triatoma		Chagas disease	

Arthropods Arthropod borne disease			
1. Mosquito	1. AnophelesMalaria2. CulexJapanese encephalitisWest Nile feverBancroftian filariasisViral arthritisViral arthritis		
	3. Aedes	Yellow fever Dengue &Dengue haemorrhagic Fever Chikungunya fever Rift valley fever	
	4. Mansonoides	Brugian filariasis	
2. Sand fly	Kala-azar Oriental sore Oroya fever Sandfly fever		
3. Tse-tse fly	Sleeping sickness		
4. Louse	Epidemic typhus, relapsing fever, trench fever, pediculosis		
5. Rat flea	Bubonic plague, endemic plague, chiggerosis, hymenolepis diminuta		
6. Black fly Onchocerciasis			
7. Reduvid bug	. Reduvid bug Chaga's disease		
8. Hard tick	d tick Tick typhus, viral encephalitis, viral haem, Fever KED, Turalemia,		
	paralysis, human babesiosis		
9. Soft tick Q fever, relapsing fever			
10. Trombiculid mite	Scrub typhus, Ricketssial pox		
11. Itch mite	Scabies		
12. Cyclops Guinea warm disease, Fish tape worm(D, Latum)		worm(D, Latum)	

INCUBATION PERIODS

Disease	Incubation period
Short-incubation	(< 1 week)
Cholera	Hours-5 days
Scarlet fever	1-3 days
Bacillary dysentery	1-7 days

Anthrax	2-5 days
Diphtheria	3-4 days
Gonorrhoea	3-4 days
Meningococcaemia	3-4 days
Intermediate incubation	(1-3 weeks)
Whooping cough	7-10 days
Poliomyelitis	
Typhus fever	7-14 days
Lassa fever	
Measles	
Typhoid	7-21 days
Mumps	14-21 days
Chickenpox	
Rubella	
Trypanosoma	
Rhodesiense	
Malaria	1 week- months
Amoebiasis	2 weeks- months
Rubella	4-14 days
Long Incubation period	(> 3 weeks)
Brucellosis	Days- months
Hepatitis A	2-6 weeks
Hepatitis B	6 weeks- 6 months
Leprosy	2-5 years
Rabies	Variable (2-8 weeks)

DAY OF APPEARING RASH AFTER FEVER

1 st	Varicella, Rubella	Very
2 nd	Scarlet fever	Sick
3 rd	Small pox	Person
4 th	Measles	Must
5 th	Typhus	Take
6 th	Dengue	Double
7 th	Typhoid	Treatment

PERIOD OF INFECTIVITY

Disease	Period of infectivity
Measles	From prodrome until 4 days after onset of rash
Mumps	3 days pre-parotitis until 1 week after.
Chickenpox, rubella	One week before onset of rash & 1 week after last crop of rash.
Diphtheria, scarlet fever	From onset to 3 weeks after.
Hepatitis A	Prior to icteric phase.
Whooping cough	7 days after exposure to weeks after onset of symptoms.

All rights reserved @ similima